DISSERTATION ON

CLINICAL PROFILE OF BABIES BORN TO PIH MOTHERS IN GOVERNMENT THENI MEDICAL COLLEGE HOSPITAL

Dissertation submitted to

THE TAMILNADU DR. M. G. R. MEDICAL UNIVERSITY

In partial fulfillment of the regulations
for the award of the degree of

M.D. DEGREE IN PEDIATRIC MEDICINE
BRANCH – VII

GOVERNMENT THENI MEDICAL COLLEGE
THENI – 635531

THE TAMILNADU DR. M. G. R. MEDICAL UNIVERSITY
CHENNAI – 32

MAY 2019

CERTIFICATE

This is to certify that the Dissertation entitled **CLINICAL PROFILE OF BABIES BORN TO PIH MOTHERS IN GOVERNMENT THENI MEDICAL COLLEGE HOSPITAL** is a bonafide record of work done by **Dr. S. IGNATIUS JOEAL DEEPTHY,** in the Department of Pediatrics, Government Theni Medical College ,Theni, during her Post Graduate Course from 2016 to 2019. This is submitted as partial fulfilment for the requirement of **M.D.,** Degree examinations – Branch-VII(Pediatrics) to be held in May 2019.

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ETHICAL COMMITTEE CERTIFICATE

Name of the Candidate : Dr. S. IGNATIUS JOEAL DEEPTHY

Course : M.D., PEDIATRICS

Period of Study : OCTOBER 2017- SEPTEMBER 2018

College : GOVERNMENT THENI MEDICAL COLLEGE

Dissertation Topic : CLINICAL PROFILE OF BABIES BORN TO PIH MOTHERS IN GOVERNMENT THENI MEDICAL COLLEGE HOSPITAL

The Ethical Committee, Government Theni Medical College has decided to inform that your Dissertation Topic is accecpted and you are permitted to proceed with the above study.

PLACE: Theni Secretary

DATE: Ethical Committee

Ref. No. 5292 /ME1/17

Government Theni Medical College
Theni Dated: 16.10.2017

Institutional Ethical Committee:

Convenor:

Dr. T. Thirunavukkarasu, M.D., D.A.,
Dean
Govt. Theni Medical College
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Sub: Medical Education – Govt. Theni Medical College,
Theni – Ethical Committee – Minutes – Communicated – Reg.

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The Ethical Committee Meeting of the Govt. Theni Medical College, Theni was held at 11.00 A.M. on 16.10.2017 at Conference Hall, Near Dean's Chamber, Government Theni Medical College, Theni.

The following Members of the Committee have attended the Meeting.

1.	**Convener**	:	**Dr. T. Thirunavukkarasu, M.D., D.A., Dean**
2.	**Member Secretary**	:	**Dr. M. Ilangovan, M.S., Deputy Superintendent**
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7.	**Public**	:	**Mr. P. Deenadhayalan, M.A.,** Land Lord, Koduvilarpatti, Theni District.

The following Project was approved by the Committee:

Name of the PG	Course	Name of the Project	Remarks
Dr. Ignatius Joeal Deepthy	MD (Paed.) II Year GTMC, Theni	Clinical Profile of Babies born to PIH Mothers in Govt. Theni Medical College Hospital, Theni	Approved

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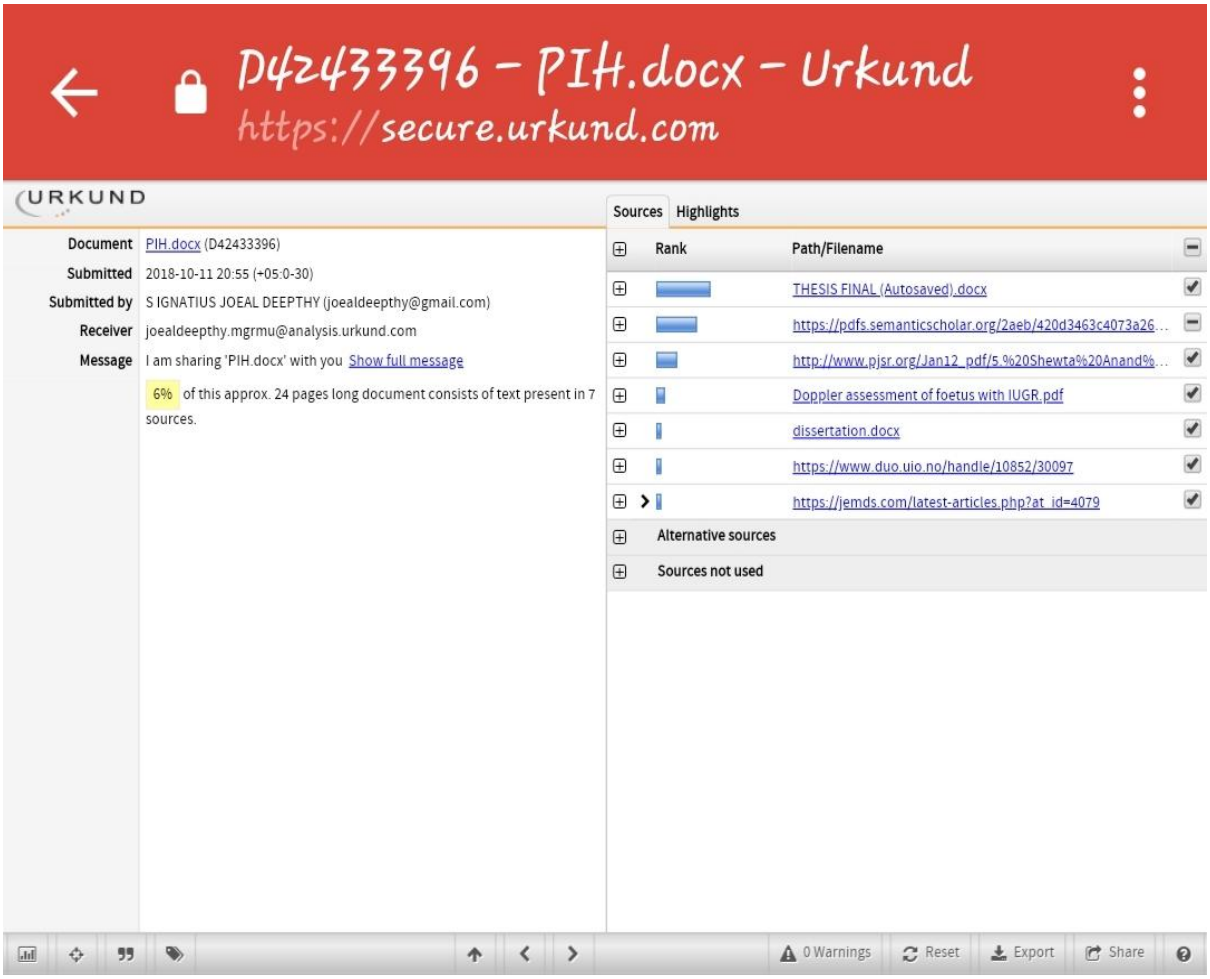
The above individual – through Head of the Department concerned.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DECLARATION

I, Dr. S. IGNATIUS JOEAL DEEPTHY, solemnly declare that the Dissertation titled **"CLINICAL PROFILE OF BABIES BORN TO PIH MOTHERS IN GOVERNMENT THENI MEDICAL COLLEGE HOSPITAL"**is a bonafide work done by me in the Department of Pediatrics, Government Theni Medical College Hospital, Theni, during the period October 2017 – September 2018.

The Dissertation is submitted to **"The Tamilnadu Dr. M.G.R. Medical University, Chennai"**, Tamilnadu as a part of fulfillment for the requirement of **M.D**.Degree examinations-Branch-VII(Pediatrics) to be held in May 2018.

Place: Theni

Date:

Dr. S. Ignatius Joeal Deepthy

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TABLE OF CONTENTS

INTRODUCTION

Hypertensive disorders in pregnancy are a major cause of maternal and perinatal morbidity and mortality[1]. Babies born to mothers with pregnancy induced hypertension are susceptible to mortality and morbidity because of immaturity, dysmaturity, physical disorders or complications during or after birth.The infants of hypertensive mothers have a significantly higher incidence of somatic growth retardation, low apgar scores, delayed adaptation, leucopenia and thrombocytopenia[2].

The most perilous time for the fetus is around the time of birth- the perinatal period, which includes both stillbirths and deaths in the first week after birth. Very early deaths are most often the sequel of events in utero and during birth, rather than resulting from the external environment into which the baby is born and thus inextricably linked to the mother's health and health care. Weight at birth, dependant almost entirely on maternal factors, is the single most important factor determining survival and health development of babies[3].

Preterm birth is a common complication of infants born to hypertensive mothers, either due to spontaneous onset of labour or to the obstetric conduct of interrupting the pregnancy due to the compromised maternal-fetal health. Prematurity increases perinatal mortality and morbidity rates with immediate or late sequelae[4].

Fetal mortality markedly increases with rising maternal diastolic blood pressure and proteinuria. Diastolic blood pressures more than 95 mm Hg are associated with a threefold rise in the fetal death rate. Fetal morbidity may include IUGR, fetal acidemia and complications from prematurity. Approximately one-third of the infants born to mothers with hypertensive disorders have decreased platelet count at birth, but the counts generally increase rapidly to normal levels. 40%-50% of newborns have neutropenia that generally resolves before three days of age. These infants may be at an increased risk of neonatal infections[5].

Haematological abnormalities in infants born to hypertensive mothers can lead to serious neonatal complications like sepsis, increased predisposition to infections and disseminated intravascular coagulation(higher in preterm than in term neonates). Bleeding manifestations including intracranial haemorrhage may result from platelet deficiency due to any cause[6].

The classification of the hypertensive disorders complicating pregnancy by the Working Group of the National High Blood Pressure Education Program(NHBPEP)[2000] is as follows. There are four types of hypertensive diseases.

1) Chronic hypertension

2) Preeclampsia-Eclampsia

3) Preeclampsia superimposed on chronic hypertension

4) Gestational hypertension[7]

Chronic hypertension

Blood pressure more than or equal to 140/90 mm Hg before pregnancy or diagnosed before 20 weeks of gestation

Or

Hypertension first diagnosed in pregnancy and persists after 12 weeks postpartum

Preeclampsia-Eclampsia

This pregnancy specific syndrome usually occurs after 20 weeks of gestation.

Preeclampsia

Minimum criteria

BP more than or equal to140/90 mm Hg after 20 weeks gestation

Proteinuria more than or equal to 300mg/24 hours or more than or equal to 1+ dipstick

Increased certainty of preeclampsia

BP which is more than or equal to 160/110 mm Hg

Proteinuria 2.0g/24hrs (or) more than or equal to 2+ dipstick (The proteinuria should occur for the first time in pregnancy and regress after delivery)

Serum creatinine > 1.2mg/dl (unless known to be previously elevated)

Platelets < 1,00,000/mm^3 and/or evidence of microangiopathic haemolytic anaemia

Elevated hepatic enzymes (ALT or AST)

Persistent headache or other visual or cerebral disturbances

Persistent epigastric pain

Eclampsia

Seizures which cannot be attributed to any other cause in a woman with preeclampsia

Preeclampsia superimposed on chronic hypertension

New onset proteinuria more than or equal to 300mg/24 hours in women with hypertension but no proteinuria before 20 weeks gestation

Sudden increase in proteinuria or blood pressure or platelet <1,00,000/mm^3 in women with hypertension and proteinuria even before 20 weeks of gestation

Abnormal increase in ALT or AST

Gestational hypertension

Blood pressure more than or equal to 140/90 mmHg for the first time during pregnancy

No proteinuria

BP returns to normal before 12 weeks postpartum

Final diagnosis is made only postpartum

Women with the preeclampsia syndrome who have not yet manifested proteinuria and women who do not have the syndrome.

The clinical importance of making such a distinction is that preeclampsia is associated with fetal risks such as intrauterine growth restriction, prematurity and death; while hypertension without proteinuria generally has a far more benign course.

Preeclampsia is believed to be a 2 stage disease with shallow cytotrophoblastic invasion of maternal spiral arterioles initially resulting in placental insufficiency. Acute or chronic uteroplacental insufficiency results in intrapartum or antepartum anoxia that may lead to preterm delivery, IUGR and fetal death. Prematurity is one of the most important factor responsible for increased perinatal mortality and morbidity. Complications occurring in babies of preeclamptic mothers closely relates to the severity of proteinuria and hypertension.

Preeclampsia is associated with adaptive changes in the fetal circulation and the placentally derived factors implicated in the pathogenesis of maternal manifestations of the disease are known to contribute to the development of growth restriction and neonatal thrombocytopenia. Severe hypertension causes a marked imbalance in the homeostatic system of the baby.

These babies of the hypertensive mothers may have a spectrum of haematological changes which may add on to the existing morbidity in them. Occurrence of severe neonatal thrombocytopenia was reported to be significantly associated with prematurity and low birth weight. Preterm and low birth weight babies born to mothers with gestational hypertension, preeclampsia

and eclampsia would require follow-up for thrombocytopenia in the early days of neonatal period[8].

Neonatal outcome is influenced by gestational age and the severity of hypertension as expressed by the need for antihypertensive treatment. Severe preeclampsia is associated with different degrees of fetal injury. The major impact on the fetus is under nutrition as a result of uteroplacental insufficiency, which can lead to growth retardation. There are long and short term effects. The short term effect observed is altered fetal growth resulting in greater fetal liability. The fetal health as well as its weight is highly compromised, leading to various degrees of fetal morbidity and fetal damage[9].

The consequences of babies born to hypertensive mothers are detailed as follows.

PREMATURITY

Live born infants delivered before 37 weeks from the first day of LMP are designated as premature by WHO(World Health Organisation). Most preterm births are spontaneous without an identifiable cause. Prematurity can be due to innumerable causes such as multiple gestation, placenta previa, abruption placenta, preeclampsia, chronic medical illness, intrauterine infection, maternal drug abuse etc. Of these preeclampsia contributes to a significant proportion of premature infants.

Prematurity increases the severity but decreases the distinctiveness of the clinical presentation of most neonatal diseases. Immature functioning of the organ systems, complications of therapy and the specific disorder that has lead to the premature onset of labor contribute to the neonatal mortality and morbidity.

Conventionally gestational age of neonates is computed based on Naegele's formula or by ultrasonic evaluation during pregnancy, or after birth, using the New Ballard assessment and scoring. Gestational age estimates based on Naegele's formula have lower accuracy in settings with low literacy and are likely to be affected by variation in ovulation and also by breastfeeding. Ultrasound, as a tool to assess gestational age, is a limiting factor, since not all women undergo regular ultrasonic evaluation during pregnancy, in developing countries like India. Assessment of gestational age by New Ballard Score(NBS) and its accuracy depends on the skill of the examiner and the condition of the neonate.

The Ballard score is based on the neonate's physical and neuromuscular maturity and can be used upto 4 days after birth. It assigns a score to various criteria, the sum of all of which is then extrapolated to the gestational age of the fetus. The neuromuscular components are more consistent over time because the physical components mature quickly after birth. However the neuromuscular components can be affected by illness or maternal drugs. The Ballard score is accurate only within plus or minus 2 weeks[10].

The New Ballard Score www.ballardscore.com

NEUROMUSCULAR MATURITY

SIGN	SCORE							SIGN SCORE
	-1	0	1	2	3	4	5	
Posture								
Square Window	 >90°	 90°	 60°	 45°	 30°	 0°		
Arm Recoil		 180°	 140°-180°	 110°-140°	 90°-110°	 <90°		
Popliteal Angle	 180°	 160°	 140°	 120°	 100°	 90°	 <90°	
Scarf Sign								
Heel To Ear								
TOTAL NEUROMUSCULAR SCORE								

MATURITY RATING

TOTAL SCORE	WEEKS
-10	20
-5	22
0	24
5	26
10	28
15	30
20	32
25	34
30	36
35	38
40	40
45	42
50	44

SIGN	SCORE							SIGN SCORE
	-1	0	1	2	3	4	5	
Skin	Sticky, friable, transparent	gelatinous, red, translucent	smooth pink, visible veins	superficial peeling &/or rash, few veins	cracking, pale areas, rare veins	parchment, deep cracking, no vessels	leathery, cracked, wrinkled	
Lanugo	none	sparse	abundant	thinning	bald areas	mostly bald		
Plantar Surface	heel-toe 40-50mm: -1 <40mm: -2	>50 mm no crease	faint red marks	anterior transverse crease only	creases ant. 2/3	creases over entire sole		
Breast	imperceptable	barely perceptable	flat areola no bud	stippled areola 1-2 mm bud	raised areola 3-4 mm bud	full areola 5-10 mm bud		
Eye / Ear	lids fused loosely: -1 tightly: -2	lids open pinna flat stays folded	sl. curved pinna; soft; slow recoil	well-curved pinna; soft but ready recoil	formed & firm instant recoil	thick cartilage ear stiff		
Genitals (Male)	scrotum flat, smooth	scrotum empty, faint rugae	testes in upper canal, rare rugae	testes descending, few rugae	testes down, good rugae	testes pendulous, deep rugae		
Genitals (Female)	clitoris prominent & labia flat	prominent clitoris & small labia minora	prominent clitoris & enlarging minora	majora & minora equally prominent	majora large, minora small	majora cover clitoris & minora		
TOTAL PHYSICAL MATURITY SCORE								

Gestation by Dates

	weeks

Birth date	Hour	
		am pm

APGAR	1 min	5min

Scoring

Gest. Age by Maturity Rating	_____weeks
Time of Exam	Date_____ am Hour_____ pm
Age at Exam	_____hours

Signature of Examiner

M.D. / R.N.

Neonatal problems associated with premature infants are as follows

RESPIRATORY

Hyaline Membrane Disease (HMD) or Respiratory Distress Syndrome (RDS)

Bronchopulmonary Dysplasia(BPD)

Pneumothorax

Congenital Pneumonia

Interstitial emphysema

Apnea

CARDIOVASCULAR

Patent Ductus Arteriosus(PDA)

Hypotension

HEMATOLOGIC

Anaemia

GASTROINTESTINAL

Necrotizing Enterocolitis(NEC)

Poor Gastrointestinal Motility

Hyperbilirubinemia

METABOLIC-ENDOCRINE

Hypoglycemia/Hyperglycemia

Hypocalcaemia

Hypothermia

- Metabolic acidosis
- Osteopenia

CENTRAL NERVOUS SYSTEM

- Intraventricular Hemorrhage
- Periventricular Leukomalacia
- Seizures
- Hypotonia
- Retinopathy of Prematurity
- Deafness

RENAL

- Hyponatremia/Hypernatremia
- Hyperkalemia
- Renal tubular acidosis
- Renal glycosuria

INFECTIONS

- Congenital
- Perinatal
- Nosocomial

INTRAUTERINE GROWTH RETARDATION

Intrauterine Growth Retardation is defined as the rate of fetal growth that is less than normal for the growth potential of the infant and for the population. Insufficient endometrial surface for placental invasion and growth, along with abnormal placental perfusion, may combine to restrict nutrient delivery to the fetus, leading to IUGR. Poor placental growth and function limit placental supply of growth promoting hormones to the fetus such as hPL, steroid hormones and IGF-1 and limit effective maternal-fetal nutrient exchange[11,12]. Preeclamptic mothers have poor endometrial vascular support for growth of the placenta, hence leading to placental growth failure, fetal nutrient deficit and IUGR[13]. Fetal hypoxemia, acidosis and hypoglycaemia are usually present in such cases of poor placental development and perfusion. These factors lead to increased production of prostaglandins and the activation of labour promoting cytokines, leading to preterm delivery[14].

Placental and fetal growth both depends on an adequate supply of maternal blood to the placenta. Inadequate development of the uteroplacental circulation is associated with IUGR. Radioisotope studies have demonstrated more than a twofold blood flow reduction in these pregnancies compared to normal pregnancies[15]. IUGR in the second half of gestation is primarily due to a failure of the normal villous vascular tree, mostly in the phase of nonbranching angiogenesis, because the terminal villi are critical for oxygen and nutrient transport to the fetus[16]. This angiogenesis is in turn dependant on the

cytotrophoblast invasion of the uterus and its arterioles. Cytotrophoblast invasion is actually a differentiation process whereby the cells lose the ability to proliferate and modulate their expression of state-specific antigens. These antigens include members of the integrin family of cell- extracellular matrix receptors that are required for migration and invasion of the decidua and endometrium of the uterus[17].

Preeclamptic placentas have decreased growth of terminal villi, which limits glucose, aminoacid and oxygen transport to the fetus. Preeclampsia begins with shallow cytotrophoblast invasion[18]. Abnormal cytotrophoblast invasion also occurs, as evidenced by the cells inability to switch on their integrin repertoire[19]. Hence hypoxia of the invading cytotrophoblast cells increases cytotrophoblast proliferation over differentiation and invasion, thus causing a stage for deficient placental development that can result in deficient growth factor and nutrient supply to the fetus, producing fetal growth restriction.

Therefore IUGR produces infants who are Small For Gestational Age(SGA), but also infants who are Appropriate For Gestational Age(AGA) who experienced reduced fetal growth rates in utero. Small For Gestational Age(SGA) infants can be the result of normal but slower than average rates of fetal growth[20]. SGA infants are being classified as having symmetric or asymmetric IUGR. Symmetric IUGR means that both body and brain growth are limited relatively equally. Asymmetric IUGR means that body growth is

restricted to a much greater extent than the brain growth. Eventhough growth of the brain is spared relative to overall fetal growth, head circumference is mostly below the 10^{th} percentile for gestational age and there is reduced brain volume[21]. The heart is also larger for body weight and spared in these babies, whereas the thymus and liver are smaller for body weight. Contributing factor includes an increased rate of cerebral blood flow relative to the systemic and umblical circulations.

Hence, the factors intrinsic to the fetus cause symmetric growth restriction, whereas extrinsic factors cause asymmetric growth restriction. Intrinsic factors that limit the growth of both the fetal body and brain include congenital infections(toxoplasmosis, rubella, cytomegalovirus), chromosomal anomalies, some inborn errors of metabolism and some drugs. Due to their intrinsic nature, patterns of symmetric growth restriction develops during the early periods of gestation.

Asymmetric growth restriction typically develops during the late second and third trimesters. This has been due to the reduction in energy substrate supply to the fetus, thereby limiting fat and glycogen storage and the growth of skeletal muscle, but allowing for continued bone and brain growth. Extremely premature infants are often Small for Gestational Age(SGA) and have asymmetric growth, most probably due to common underlying pathology such as placental insufficiency, that produced the preterm delivery and growth restriction. More extreme limitations of nutrients for prolonged periods affect

both energy storage and growth, hence causing reductions in head circumference and length as well as soft tissue mass and body weight. With decreased nutrient supply early in gestation, growth of all body organs is restricted, whereas decreased nutrient supply later in gestation primarily restricts the growth of skeletal muscle and adipose tissue.

Intrauterine growth retardation related to impaired placental function is usually associated with increased umbilical artery impedance, typically followed by brain sparing. In the course of worsening obliteration of placental vessels, venous shunting across the ductus venosus occurs and results in an increased blood flow to the heart at the sparing of liver. The increase in right ventricular afterload causes further shunting of blood to the left ventricle that improves the left ventricular output. Increased end diastolic pressure in the right ventricle, combined with decreased cardiac compliance, is reflected as a decrease, absence or reversal of blood flow in the ductus venosus during the atrial systolic component of the waveform. Further worsening of the placental function will lead to increased central venous pressure and umbilical venous pulsations which can be seen in the Doppler ultrasound. These may lead to abnormal biophysical profile or loss of fetal heart rate variability.

The concepts of ‘fetal malnutrition’ was first developed by Clifford and was defined by Scott and Usher as a clinical state of babies characterized by obvious intrauterine loss of failure to acquire normal amount of subcutaneous

fat and muscle[22,23]. The assessment of nutrition at birth has been made using several systems:

1. Anthropometry- Weight, Length, Head and Chest circumference
2. Proportionality indices- Ponderal Index(PI), head circumference to length ratio, chest circumference or midarm circumference to head circumference ratio(MAC/HC).
3. Clinical Assessment of Nutrition(CAN) of the fetus and the score- CAN score is a scoring system based on nine 'superficial' readily detectable signs of malnutrition in the neonate.

CAN score has nine superficial readily detectable signs, which are rated 1(worst-severe FM) to 4(best well nourished). The highest possible score is 36 and lowest possible score is 9. A CAN score of < or equal to 24 was taken as fetally malnourished[24,25].

The Nine signs for CAN Status in the Newborn[24]

- **Hair** Large amount, smooth, silky, easily groomed (4).

  Thinner, some straight, 'staring' hair (3).

  Still thinner, more straight, 'staring' hair which does not respond to brushing (2).,

  Straight 'staring' hair with depigmented strip (flag sign) (1).

- **Cheeks** Progression from full buccal pads and round face (4); to significantly reduced buccal fat with narrow, flat face (1)

- **Neck and Chin** Double or triple chin fat fold, neck not evident (4); to thin chin. No fat fold, neck with loose, wrinkled skin, very evident (1).
- **Arms** Full, round, cannot elicit ‘accordion’ folds or lift folds of skin from elbow or tricep area (4); to a striking ‘accordion’ folding of lower arm, elicited when examiner’s thumb and fingers of the left hand grasps the arm just below the elbow of the baby and thumb and fingers of the examiners right hand circling the wrist of the baby are moved towards each other; skin is loose and easily grasped and pulled away from the elbow.
- **Legs** Like arms.
- **Back** Difficult to grasp and lift skin in the interscapular are (4); to skin loose, easily lifted in a thin fold from the interscapular area (1).
- **Buttocks** Full round gluteal fat pads (4); to virtually no evident gluteal fat and skin of the buttocks and upper posterior high loose and deeply wrinkled (1)
- **Chest** Full, round, ribs not seen (4); to progressively prominence of the ribs with obvious loss of intercostal tissues (1).
- **Abdomen** Full, round, no loose skin (4); to distended or scaphoid, but with very loose skin, easily lifted, wrinkled and ‘accordion’ folds demonstrable.

The perinatal problems and the central nervous system sequelae, occurred primarily in fetaly malnourished babies, whether AGA or SGA, but not those who were simply SGA but not malnourished. In utero growth restriction is not a uniform condition with respect to its severity and duration, the underlying pathogenesis and the developmental stage of the fetus at the time of its occurrence. If malnutrition happens early in the second trimester, length, weight and head circumference are all significantly reduced, whereas if length and head circumference are less affected but baby is small and underweight mostly the malnutrition happened in the beginning of the third trimester.

At the same birth weight, SGA/IUGR infants have a lower risk of neonatal deaths compared with preterm AGA infants. Compared to the AGA infants of the same gestational age, SGA/IUGR infants have a higher incidence of neonatal mortality and morbidity. In general SGA/IUGR infants and children are at higher risk for poor postnatal growth, neurological impairment, delayed cognitive development and poor academic achievement. Finally some adults who were SGA/IUGR at birth appear to have a higher risk of coronary heart disease, hypertension, non insulin dependent diabetes, obstructive pulmonary disease, renal impairment, decreased reproductive function as well as other health risks and growth related psychosocial issues.

Ponderal Index can be used to identify infants whose soft tissue mass in less than the normal for the stage of skeletal maturity. This index is independent of gender, race, birth order and to a certain extent, gestational age. It has been used as an indicator of fetal growth status, specifically to assess asymmetrical intrauterine growth retardation.

$$\text{Ponderal Index(PI)} = \frac{\text{Birth weight(gms)} \times 100}{(\text{Crown heel length in cm})^3}$$

PONDERAL INDEX	SIGNIFICANCE IN NEWBORN
>2.5	Term, appropriate for age
<2	Asymmetrical IUGR
>2	Symmetrical IUGR

The inutero PI also proved to be a valuable index in the prediction of fetal outcome in those cases of IUGR in whom the in utero PI was smaller than 1 SD from the average. Fetal and neonatal well being was clearly compromised when IUGR was associated with a low inutero PI. Cesarean section delivery and fetal distress rates were significantly higher for infants with a low birth weight. PI appears to be a better measure of infants with IUGR problems than birth weight percentile.

A prevailing hypothesis regarding the pathogenesis of preeclampsia is the 'ischemic model'. Decreased uteroplacental perfusion is hypothesized to be the primary step and the point of convergence of diverse pathogenic processes in the development of preeclampsia[26,27,28]. It is intuitive that reduced placental blood flow should result in decreased fetal growth, with an increased risk of intrauterine growth restriction and low birth weight.

There has also been limitations to the 'ischemic model' of preeclampsia. The considerable pathophysiologic heterogeneity in preeclampsia is also identified[29]. Studies have demonstrated an increase in blood flow among preeclamptic patients secondary to increased maternal cardiac output[30]. By studying the placental clearance of dehydroisoandrosterone sulphate, an indicator of uteroplacental perfusion, the women who became preeclamptic in the third trimester had substantially greater uteroplacental blood flows throughout most of their pregnancies than those who remained normotensive[31].

Midarm circumference/ Head circumference ratio, independent of birth weight readily discriminated the late gestation growth retarded baby. This test can be used as a reliable test to identify neonates whose growth is retarded, even when their weight does not fall below 10^{th} percentile. But those babies whose head circumference is reduced because of proportionate growth retardation might not be identified[32].

LOW APGAR SCORES

The APGAR score was devised by Dr.Virginia Apgar in 1952. It is a quick method of assessing the clinical status of the newborn infant. The APGAR score comprises of five components[33]

- Heart rate
- Respiratory effort
- Muscle tone
- Reflex irritability
- Colour

Each component is given a score of zero, one or two. It is important to recognize that these elements of APGAR score are partially dependant on the physiologic maturity of the infant. A low APGAR score may be the result of fetal distress but it can also be caused by a number of factors including preterm delivery and drugs given to the mother during labour. The APGAR score was not designed to predict neurological outcome. It is generally performed at 1 minute and again at 5 minutes after birth. However, the resuscitative measures must be initiated before the 1-minute score is assigned. Therefore, it is not used to guide the resuscitation. If the 5 minute score is 6 or less, the scoring is then repeated at successive 5-minute intervals until it is >6.

ONE MINUTE APGAR: It generally correlates with umbilical cord blood pH and is an index of intrapartum depression. It usually does not correlate with the outcome. Neonates with a score of 0 to 4 have been shown to have a

significantly lower pH, higher partial pressure of carbon di oxide($PaCO_2$) and lower buffer base than those with APGAR scores >7.

APGAR SCORES BEYOND 1 MINUTE: They are reflective of the infant's changing condition and the adequacy of resuscitative efforts. Persistence of low APGAR scores indicates need for further therapeutic efforts and usually the severity of the neonate's underlying problem. The more prolonged the period of depression, the more likely is an abnormal long term neurologic outcome.

Low APGAR scores may be indicative of numerous maternal and fetal factors. One of them is the presence of pregnancy induced hypertension in the mothers. The presence of increased blood pressure and proteinuria in preeclampsia are associated with low fetal birth weight and lower APGAR score and an increased risk of adverse perinatal outcome. In cases of severe preeclampsia, the APGAR score at one minute is two fold worse than in mild preeclampsia

In women with preeclampsia, there is insufficient placental circulation. The association between preeclampsia and abnormal placentation is known to involve in trophoblast invasion of maternal spinal arteries. Abnormal placentation results in inadequate uteroplacental blood flow that can lead to unsuccessful pregnancy outcomes. Low APGAR scores indicate the adverse state of the newborn. Low APGAR scores most commonly results from uteroplacental insufficiency which is a later clinical manifestation of poor

placentation and placental ischaemia as caused by preeclampsia. This may result in perinatal mortality and morbidity.

FETAL ORIGINS OF ADULT DISEASE

Inutero development is characterized by a state of rapid cellular and molecular growth. The ontological processes which are critical for maturation of the fetus are highly sensitive to alterations in the intrauterine environment[34]. The insults from preeclampsia exposure accured during the sensitive periods of development may predispose these babies to an increased risk of disease states such as hypertension, obesity and diabetes in adulthood[35]. This concept underscores the fact that the physiologically immature fetus is highly susceptible to disruptions in the inutero placental blood flow that results from preeclampsia.

There have been long term followup studies which have demonstrated that babies who have suffered intrauterine growth retardation are more likely to develop hypertension, coronary artery disease and diabetes in adult life. Unusual perinatal complications involving anoxia or catecholamine release in the fetus or newborn may predispose these babies to the development of precocious coronary atherosclerosis in later life[36].

Many of these fetuses have to adapt to a limited supply of nutrients. In doing so, these fetuses permanently change their structure and metabolism. These ‘programmed’ changes may be an option for the origin of few diseases in

later life, including coronary heart disease and related disorders such as stroke, diabetes and hypertension.

Babies who are growth retarded at birth or who have altered placental growth are known to have increased rates of hypertension, coronary heart disease and Type II diabetes mellitus in adult life. These associations are thought to result from fetal programming, whereby an insult or stimulus at a critical and sensitive period of early life has permanent effects on the body's physiology, structure and metabolism. Smaller size at birth and disproportionate head size, weight and length appear to be surrogate markers for the actual influences that programme the fetus. Adult cardiovascular disease may be a consequence of fetal adaptations invoked when the materno-placental nutrient supply fails to match the fetal nutrient demand.

Studies have demonstrated that IUGR babies, as defined by a birth weight below the 10^{th} percentile, gives rise to a reduction in the nephron numbers. Oligonephropathy has been suggested to substantially increase the risk for systemic and glomerular hypertension in adult life as well as enhanced risk for expression of renal disease after exposure to potentially injurious renal stimuli.

In the setting of severe preeclampsia the risk of fetal death outweighs the potential benefits of pregnancy prolongation. However, in mild preeclampsia, the risk of fetal demise is over 50% less than pregnancies with severe preeclampsia. Despite paucity of data to guide the clinical decision making in pregnancies with mild preeclampsia, obstetricians have to balance the small risk

of fetal demise with the benefits of pregnancy prolongation and potential for the continued inutero maturation, more particularly in pregnancies less than 37 weeks gestation[37].

Preeclampsia, which is a condition characterized by decreased uteroplacental blood flow and ischaemia, is definitely a significant risk factor in the development of IUGR and represents the most common cause of IUGR in the nonanamolous infant. Pregnancies complicated by severe preeclampsia had infant birth weights 12% lower than expected, while mild preeclampsia pregnancies showed no significant difference in weight gain from expected norms[38].

HEMATOLOGICAL EFFECTS

Maternal preeclampsia can result in neonatal thrombocytopenia which is typically defined as a platelet count<1,50,000/microliter[39].The degree of severity of thrombocytopenia has been subcategorized according to platelet count as follows: Mild thrombocytopenia-platelet count 100,000 to 150,000/microlitre; moderate thrombocytopenia-platelet count 50,000 to 99,000/microlitre; severe thrombocytopenia-platelet count <50,000/microlitre. In preeclamptic pregnancies, thrombocytopenia is usually identified at birth or within the first 2-3 days of life, with most cases resolving by 10 days of life[40]. The severity of thrombocytopenia related to preeclampsia is highly variable,

which is evident by a small percentage of babies developing clinically significant or severe thromobocytopenia(<50,000/microliter)[41,42]

Platelets are tiny cellular fragments produced by megakaryocytes in the bone marrow. Platelet production, or thrombopoiesis, is a complex process that results in the production of thrombopoietin as the thrombopoietic stimulus leading to the generation and proliferation of magekaryocyte progenitors. The pathogenesis of thrombocytopenia among babies born to preeclamptic mothers is unknown. One mechanism is that preeclampsia and the resultant fetal hypoxia has a direct suppressant effect on megakaryocyte proliferation[43].This is supported by studies that show IUGR neonates have significant megakaryocytopoietic defects without evidence of increased platelet destruction[44]. Another causative mechanism could be due to increased platelet activation mediated through cytokines, thrombopoietin and interleukin-6[45]. It has also been studied that there is a common precursor cell for erythrocytic and megakaryocytic cell lines. So chronic exposure to increased levels of erythropoietin in the fetus due to fetal hypoxia may also lead to thrombocytopenia by suppressing the megakaryocytic cell line which may lead to decreased platelet production. Thrombocytopenia could also occur as a result of thrombocyte adherence to the damaged endothelial region caused by segmental vasospasm and vasodilatation in the placenta of hypertensive mothers. Abnormal placental endothelial surface caused thrombocyte

destruction and the resulting thrombocytopenia improved in a short time after delivery.

In addition to the effects of preeclampsia on platelets, these neonates have a 50% incidence of neutropenia(defined as an absolute neutrophil count<500)[46].Neutropenia due to PIH is the most common type of neutropenia seen in the neonatal intensive care unit. The ANC count can be very low, more frequently below 500/microlitre, but the count generally spontaneously rises within the first few days and is almost always greater than 1000/microlitre by day 2 or 3. Usually no leukocyte 'left shift' is seen, and no Dohle bodies, toxic granulation or vacuolization is present in the neutrophils. Hence neutropenia has a variable course, which typically lasts days to weeks in the affected infants.

The biological mechanism for neonatal neutropenia resulting from preeclampsia has not been fully elucidated. One mechanism is that preeclampsia and the resultant uteroplacental insufficiency inhibits fetal bone marrow production of the myeloid lineage which manifests by a decrease in neutrophil count[40]. Another mechanism proposed to be contributory to the pathogenesis of neutropenia in neonates of hypertensive mothers is the activation of the Fas/Fas ligand pathway of apoptosis.The increased apoptotic activity of myeloid precursors may be contributory to the pathogenesis of neutropenia. One another mechanism is that it is caused by an inhibitor of neutrophil production of placental origin that might function mechanistically by depressing natural G-CSF production.

Neutropenia associated with preeclampsia is also associated with reduced numbers of circulating colony forming unit-granulocyte macrophage(CFU-GM) and decreased neutrophil storage pools[47]. Neutropenia is usually self limited although in few cases it may be severe enough to warrant therapy with G-CSF(Granulocyte-Colony Stimulating Factor).

These babies of hypertensive mothers are also at risk for increased number of nucleated RBCs. Preclampsia when associated with placental hypoperfusion results in hypoxic response in developing fetus in the form of increased erythropoiesis and release of immature erythrocytes. It has been stated that the cytotrophoblasts are unable to differentiate correctly which can lead to failure of invasion of cytotrophoblasts and its arterioles into the uterus. This relatively lead to hypoxic environment in the placenta which can result in increased production of erythropoietin which in turn leads to stimulation of erythropoiesis and thereby increased number of nucleated RBCs. Therefore increased number of nucleated RBCs is considered as a marker of degree of intrauterine hypoxia[48].

HbF has a higher affinity for oxygen compared to HbA. The reason for this is that HbF does not interact with 2,3 diphosphoglycerate at a significant level, and cells which contain HbF display a higher oxygen affinity and have the advantage of extracting more oxygen from maternal blood by the placenta. HbF levels were found to be raised in the umbilical cord blood in preeclamptic infants.

REVIEW OF LITERATURE

A study conducted by Ravikant Patel et al in GMERS medical college, Gujarat[49] stated that 54.69% PIH mothers had preterm delivery. 53.12% of babies were of low birth weight and 7.81% of babies were intrauterine growth retarded babies. 18.75% of babies required NICU admission for various reasons with 1.56% neonatal death.

Shweta Anand[50] et al conducted a study in Chirayu Medical College and Research, Bhopal and concluded that the incidence of small for gestational age(SGA) was four times more in the hypertensive mothers compared to normotensive mothers. The study further revealed that 60% babies were preterm and 44.5% were small for gestational age(SGA) in the study group containing hypertensive mothers. Mean birth weight of babies born to PIH mothers was 1.7kg. Preterm IUGR were 57.1% with 71% of babies being asymmetrical IUGR. 75.5% babies required hospitalisation in the NICU.

In a study conducted by Sulaeman A Susilo[51] et al in Jakarta, Indonesia on 446 preeclamptic women stated that 19%(86/446) and 5.4%(24/446) of neonates had low APGAR scores at 1 minute and 5 minutes respectively.

Abdul karem Jasem Mohammed et al[52] conducted a study on 'The effect of pregnancy induced hypertension on Complete Blood Count of newborn' in Baghdad, Iraq and concluded that caesarean section was the major mode of delivery in the hypertensive mothers. Preterm delivery was 64% in the hypertensive mothers. The mean birth weight of these babies of PIH mothers was 2.56kg. 40% of the babies were of small for gestational age. The APGAR score at 5 minutes was significantly lower in the babies of hypertensive mothers. Thrombocytopenia was found in 50% of neonates of preeclamptic mothers.

Study conducted by Vikram Singhal et al[53] in the department of Pediatrics, Kasturba Medical College, Mangalore assessed the fetal malnutrition with CAN score and compared with weight for gestational age and Ponderal Index(PI). The study concluded that CAN score can identify fetal malnourishment in those neonates which are missed by other methods.

In another study conducted by Sandhya Sivakumar et al[54] in the department of Pediatrics, Jawaharlal Institute of Post Graduate Medical Education And Research, Pondicherry from August 2003 April 2005 concluded that the platelet counts in infants born to hypertensive mothers was significantly lower. The number of nucleated RBCs seen in the peripheral smear of the babies born to hypertensive mothers was significantly different from those

babies born to normotensive mothers. Polychromatic cells and target cells were also seen more in the peripheral smear picture of those babies born to mothers with PIH. They also stated that there was higher number of preterm, Intra Uterine Growth Restriction(IUGR) and Small for Gestational Age(SGA) babies among the infants of hypertensive mothers.

One another study conducted at R.L.Jalappa Hospital and Research Centre, Karnataka by Kalavakuru Mouna et al[55] for the duration of six months from March 2016 to November 2016 stated that 70% of babies born to preeclamptic mothers were premature and 50% of the babies developed sepsis. It also stated that the mean haemoglobin, PCV, red cell count, Mean Corpuscular Volume(MCV), reticulocyte count and nucleated RBC were significantly increased, whereas total leucocyte count, mean neutrophil count, absolute neutrophil count, lymphocyte count and platelet count were significantly decreased in babies born to preeclamptic mothers.

Manisha Shinde Jotwar[56] concluded in a study conducted at Solapur, Maharashtra that 28% of the term PIH mothers newborn were placed in NICU immediately after birth. The average birth weight of the term newborns delivered to a mother with pregnancy induced hypertension is 2400 grams.

In a study conducted by Sikha Maria Siromani[57] et al on 'Neonatal Outcome In Pregnancy Induced Hypertensive Mothers-A Tertiary Care Centre Experience' at Niloufer Hospital,Hyderabad concluded that 70.67% of mothers with PIH delivered their babies through caesarean section. 54.67% of the babies born to PIH mothers were of low birth weight. The proportion of prematurity in PIH babies was 63.01%. 15.06% of the babies born to PIH mothers had birth asphyxia. Other complications such as Transient Tachypnea of Newborn(TTN), Meconium Aspiration Syndrome(MAS) and Sepsis accounted for about 2.74%, 2.74% and 2.74% respectively. The need for NICU admission remained to be 34.25% in the babies born to PIH mothers. The APGAR score was less than 8 in 32.8% of babies born to hypertensive mothers.

In another study conducted in the year 2013 by Kwame Adu-Bonsaffoh[58] et al in Ghana stated that 78.3% of hypertensive women delivered at > or equal to 37 weeks; 9.5% delivered between 34 to 36 weeks and 12.2% of hypertensive women delivered before 34 weeks. Cesarean birth occurred in 45.7% of the hypertensive mothers. The frequency of low birth weight(LBW) was 24.7% in the preeclamptic mothers. Regarding APGAR scores, 34.0% and 14.9% of babies of hypertensive mothers had scores less than 7 at 1 minute and 5 minutes respectively. In this study, the major adverse perinatal outcomes determined among women with Hypertensive Disorders of Pregnancy include intrauterine growth restriction(6.3%), intrauterine fetal death(6.8%), preterm

delivery(21.7%), low birth weight(24.7%) and birth asphyxia or neonatal respiratory distress(15.2%).

In a study conducted at S.N.Medical College & H.S.K Hospital, Navanagar, Bagalkot from January 2012 to June 2012 concluded that 46.6% of babies born to preeclamptic mothers were preterm and 46.6% of the preeclamptic babies were delivered by LSCS.12% of the preeclamptic babies were <1.5kg, 48% were 1.5kg-2.5kg and 40% of the babies were >2.5kg. The APGAR score was < 7 in 38.6% of the preeclamptic babies.

Meghavini R Parmar et al[59] in their study conducted at GMERS Medical College, ESIC Hospital, Bapunagar, Ahemedabad on 110 cases of PIH concluded that 23 babies were 28-32 weeks, 34 babies were 33-36 weeks, 42 babies were 37-40 weeks and only one baby was of > 40 weeks gestational age at birth. 67 babies were delivered through vaginal delivery, 16 babies instrumental delivery and 17 babies were delivered through caesarean section. Birth weight was <2.5kg in 53 babies. Perinatal complications included IUGR(60.9%), birth asphyxia(8.7%), RDS(4.3%) and perinatal death(15.2%).

The study at MIMSR Medical College, Latur, Maharashtra by Vidya Devi D Kendre[60] during the period Januaury 2011 to December 2011 concluded that 60% of these babies were born by caesarean section. 40% of the neonates were

premature and 50% of them were intrauterine growth restricted. Neonatal resuscitation were required in 30% of these babies and neonatal deaths were about 8% in the babies born to hypertensive mothers. WBC count was less than 4000 in 30% of these babies and platelet count was less than 1.5 lakh in 50% of them.

Y.R.Bhat and Carol S. Cherian[61] concluded that thrombocytopenia occurred in 36% of neonates born to mothers with pregnancy induced hypertension and was severe in 20%. Occurence of severe neonatal thrombocytopenia was found to be significantly associated with low birth weight and prematurity.

N.Raizada et al[62] demonstrated that preterm infants born to hypertensive mothers are at significant risk for the development of thrombocytopenia; however severe thrombocytopenia with bleeding manifestations were unlikely to occur.

Mouzinho et al[63] reported that 40 to 50% of neonates studied developed neonatal neutropenia, and thay were younger and smaller than non-neutropenic neonates. Neutropenic neonates were more likely to be born to mothers with preeclampsia and eclampsia and the incidence of neonatal neutropenia was primarily among neonates < 30 weeks of gestation and < 1500gms birth weight.

Patricia et al showed that infants < 1200gms and < 32 weeks gestation and born to mothers with gestational hypertension, preeclampsia or eclampsia symdrome were associated with leucopenia, absolute neutropenia and thrombocytopenia.

Huang and Chang concluded that preeclampsia may cause significant normocytic normochromic erythrocythemia, marked anisocytosis (reflected by RDW) and severe thrombocytopenia in newborns.

SH Fraser and Di Tudehope[64] showed that neutropenia occurred only in infants whose mothers had eclampsia or preeclampsia. 10% of neutropenic infants developed nosocomial infection while 20% of the thrombocytopenic infants bled.

S Narayan et al found that significant correlation existed between decresing gestational age and alterations in all coagulation parameters. Higher incidence of prematurity, hyperbilirubinemia and significant prolongation in prothrombin time(PT), partial thromboplastin time with kaolin(PTTK) and thrombin time(TT) values were observed with increasing severity of grade of gestational hypertension. Incidence of disseminated intravascular coagulation was higher in preterm neonates than in term neonates.

Tsao et al concluded that neutropenia is commonly seen in infants born to mothers with gestational hypertension, preeclampsia or eclampsia syndrome, which in association with low gestational age were significantly associated with neutropenia in premature infants whereas very low birth weight was associated significantly with thrombocytopenia. Low G-CSF may contribute to neutropenia in infants born to these mothers.

Sandra et al found infants exposed to gestational hypertension, preeclampsia or eclampsia had a higher incidence of neutropenia than unexposed infants. No incidences of neutropenia occurred in any SGA infants in the absence of gestational hypertension, preeclampsia or eclapmsia. Increasing incidence of documented infection occurred with decreasing gestational age.

Moallem and Koenig[65] observed that neutropenia is a common hematologic disorder in the newborn intensive care unit, particularly in preterm neonates. Although its cause varies, a significant proportion of the episodes are associated with pregnancy complicated by preeclampsia.

AIM OF THE STUDY

- To estimate the effect of Pregnancy Induced Hypertension on mode of delivery, gestational age and birth weight of these babies.

- To estimate the impact of Pregnancy Induced Hypertension on the growth parameters and clinical status of the baby including need for NICU admission if any.

- To identify the deviations in haematological parameters in babies born to PIH mothers with special reference to platelet counts.

METHODOLOGY

STUDY DESIGN

Descriptive study

STUDY DURATION

The study was conducted for a total period of 12 months from October 2017 to September 2018.

STUDY POPULATION

The study was conducted in all neonates born to mothers with gestational hypertension, preeclampsia or eclampsia syndrome from Government Theni Medical College Hospital irrespective of NICU admission.

INCLUSION CRITERIA

Neonates born to mothers with

- Gestational Hypertension
- Preeclapmsia
- Eclampsia

EXCLUSION CRITERIA

- Infant of Diabetic Mothers
- Babies of mother with chronic hypertension
- Babies of mother with known systemic illness such as chronic renal problem, connective tissue disorders, thyrotoxicosis etc.
- Babies of mother who received drugs other than antihypertensive medications and IFA tablets
- Babies of TORCH positive mothers
- Multiple gestations
- Babies with congenital malformations

ETHICAL COMMITTEE APPROVAL

Approved

All neonates included in the study had the following done:

- Detailed maternal history like age, parity, immunisation status, gestational age, blood pressure recording, proteinuria and presence of seizures.
- Details of labor, mode of delivery, presence of complications if any during labor.
- Details of baby like name, date of birth, time of birth, apgar scores were noted.
- Gestational age was assessed by New Ballards scoring system with compensation for asphyxiated babies.
- Thorough clinical examination of the neonates was done within 24hours of birth (except head circumference).

  Anthropometric parameters

  - Head circumference: measured with non stretchable measuring tape as the maximum occipitofrontal circumference after 24 hours of life.
  - Chest circumference: measured as the maximum circumference of the chest at the level of the nipples.
  - Length: measured as the supine crown to heel length on an infantometer.
  - Weight

- Estimation of weight for gestational age was done using Fentons growth chart and baby identified to be SGA/IUGR or not.
- Ponderal Index was calculated and was considered to differeniate symmetrical and asymmetrical IUGR.
- Any indication for admission to NICU was recorded.
- CAN score was calculated from the nine superficial readily detectable signs.
- Neonatal outcome data with respect to asphyxia and its degree, Respiratory Distress Syndrome, Meconium Aspiration Syndrome, sepsis and neonatal death were recorded.
- Two ml of venous blood anticoagulated with EDTA was collected from the babies within the first 24 hours of birth and various haematological parameters studied. Hb, TC, DC and Platelet count were estimated using automated cell counter method. All these investigations were done at the central laboratory at Govt Theni Medical College.

STATISTICAL ANALYSIS

Data will be entered in excel sheet; Statistical analysis of the data will be performed by statistical software SPSS. Outcome variables will be categorized as normal or abnormal and their prevalence will be expressed as percentage and p value of < 0.05 will be considered significant.

ETHICAL ISSUES

Parents of all babies recruited in the study were explained about the methodology and investigations in detail and consent obtained.

RESULTS AND OBSERVATIONS

During the period of October 2017 to September 2018, a total of 357 pregnancies complicated with hypertensive disorders were conducted in Govt Theni Medical College. Out of this only 312 babies were included in our study after the inclusion and exclusion criteria are being applied.

In our study the selected 312 babies are being evaluated for the mode of delivery, birth weight, gestational age(using modified Ballards scoring system), presence and the type of IUGR(using Ponderal Index), Apgar scores at the 1 and 5^{th} minute, CANS score(to look for fetal malnutrition), need for NICU admission, presence of complications such as birth asphyxia, RDS, MAS and sepsis and also the haematological profile of the babies using complete blood count and peripheral smear.

1) MODE OF DELIVERY

Mode of delivery	Number of cases (n=312)	Percentage	Significance of LSCS
LN	96	30.8	p value-0.001 (Significant)
LSCS	216	69.2	
Total	312	100	

Table 1: Frequency and Percentage wise distribution of mode of delivery

Among the total, about 69.2% of babies were delivered by caesarean section and 30.8% were delivered by labor natural. This shows a statistical significance of caesarean section(p value-0.001) among these babies born to PIH mothers in our study.

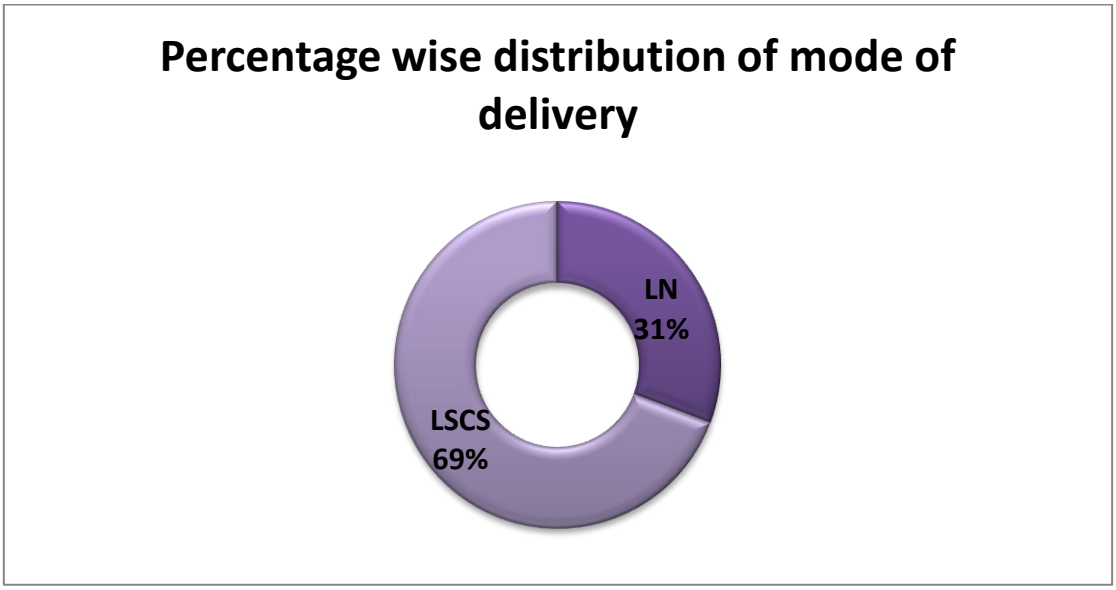

Figure 1: Percentage wise distribution of mode of delivery

MODE OF DELIVERY	LSCS	LN	TOTAL
PRETERM	127	63	190
TERM	89	33	122
TOTAL	216	96	312

Table 2: Gestation Age wise distribution of Mode of Delivery

Of the total LSCS deliveries, 127 deliveries were terminated prematurely for maternal indications and 89 babies were delivered term. Of the total LN deliveries, 63 babies were delivered prematurely and 33 babies were delivered at term.

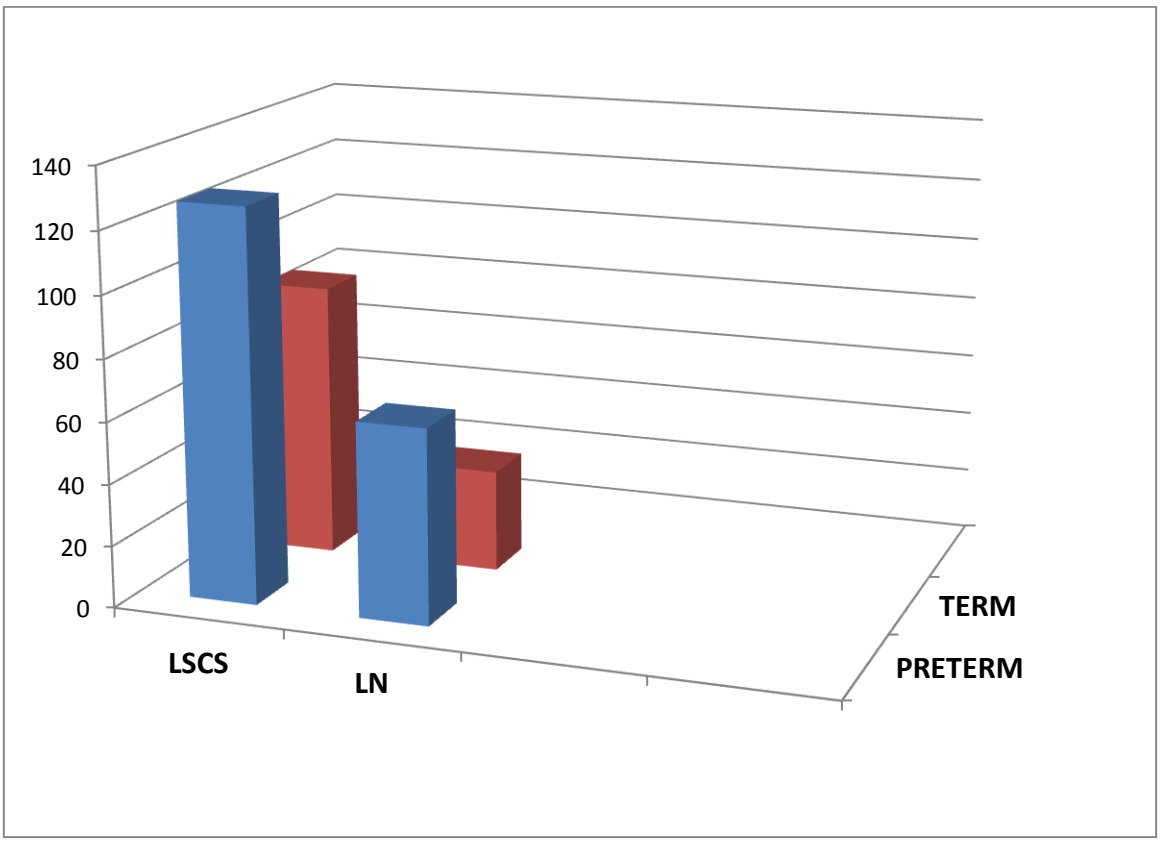

Figure 2: Gestation Age wise distribution of Mode of Delivery

2) BIRTH WEIGHT

Birth weight	Number of cases (n=312)	Percentage	Significance of LBW
<2.5 kg	215	68.9	p value-0.005 (significant)
≥2.5 kg	97	31.1	
Total	312	100	

Table 3: Frequency and Percentage wise distribution of Birth Weight

Among the total 312 babies, about 215 babies(68.9%) belong to the low birth weight category. Hence one of the most common complication of PIH babies being low birth weight(p value-0.005) is statistically significant in our study also.

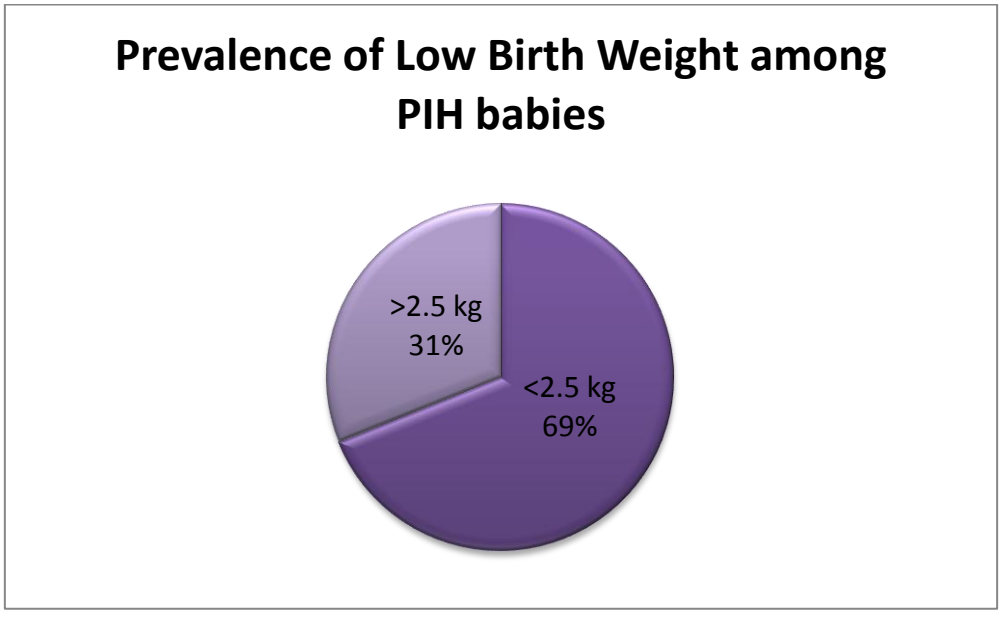

Figure 3: Percentage wise distribution of Birth Weight

Birth weight	Number of LBW babies (n=215)	Percentage
<1 kg	3	1.4
1 – 1.5	20	9.3
1.5 – 2.5	192	89.3
Total	215	100

Table 4: Weight wise distribution of Low Birth Weight babies

Among the 215 babies who are of low birth weight, 192 babies fall between 1.5-2.5kg category which contributes to a total of 89.3%.Only 20 babies are of very low birth weight(1-1.5kg) and 3 babies are of extremely low birth weight(<1kg). Thus most low birth weight babies of PIH mothers are between 1.5-2.5kg in our study.

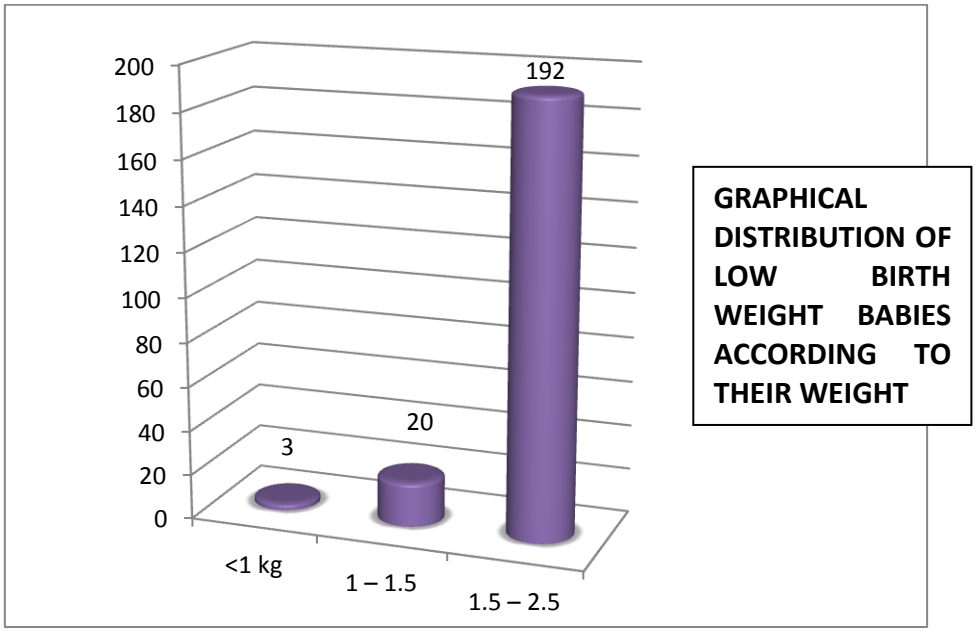

Figure 4: Weight wise distribution of Low Birth weight babies

3) GESTATIONAL AGE

Gestational Age	Number of cases (n=312)	Percentage	Significance of preterm
<37	190	60.8	p value-0.035 (significant)
≥37	122	39.2	
Total	312	100	

Table 5: Frequency and Percentage wise distribution of Gestational Age

About 60.8% of PIH babies were preterm among the total. Hence prematurity(p value-0.035) which is one another important complication of babies born to PIH mothers is statistically significant in our study.

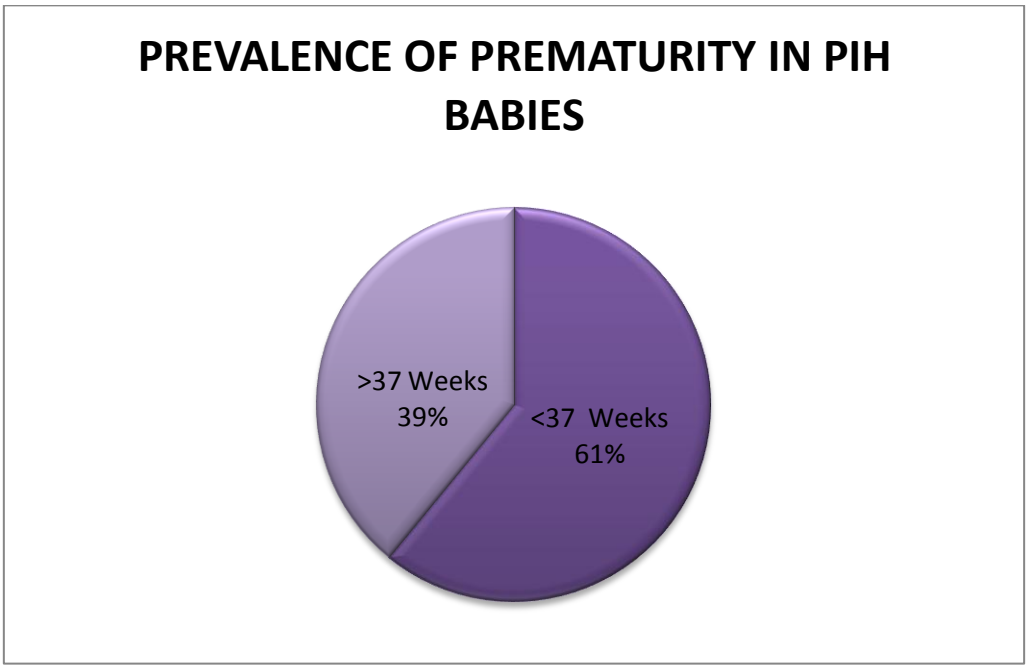

Figure 5: Percentage wise distribution of Gestational Age

Gestational Age	Number of babies (n=190)	Percentage
<35 weeks	38	20
35-37 weeks	152	80
Total	190	100

Table 6: Gestation Age wise distribution of Preterm babies

Among the 190 preterm cases, 152 babies fall under the late preterm category. Hence late preterm births represent the major subset of these PIH babies.

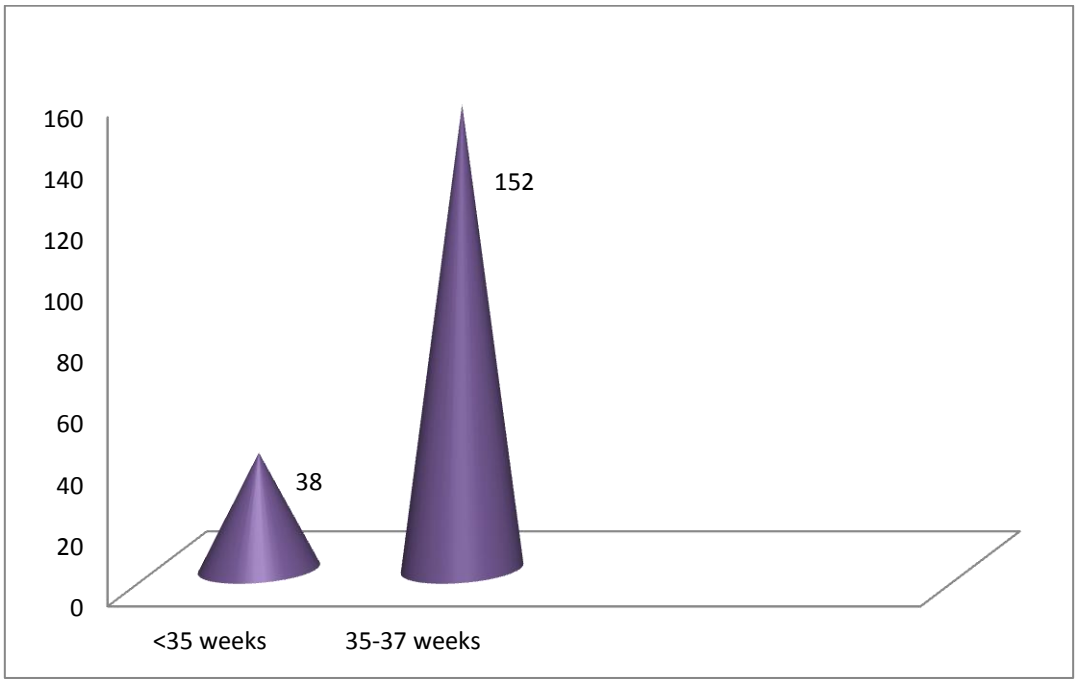

Figure 6: Gestation Age wise distribution of Preterm babies

4) SMALL FOR GESTATIONAL AGE/INTRAUTERINE GROWTH RESTRICTION

WEIGHT FOR GESTATIONAL AGE	Number of babies (n=312)	Percentage	Significance of IUGR
AGA	181	58	p value-0.042 (Significant)
SGA/IUGR	131	42	
Total	312	100	

Table 7: Frequency and Percentage wise distribution of SGA/IUGR babies

Among the total, 42% of babies were small for gestation age/intrauterine growth restricted babies which was identified by weight for gestational age < 10^{th} percentile in the Fentons chart. Also the presence of SGA/IUGR(p value-0.042) in babies of PIH mothers was statistically significant in our study.

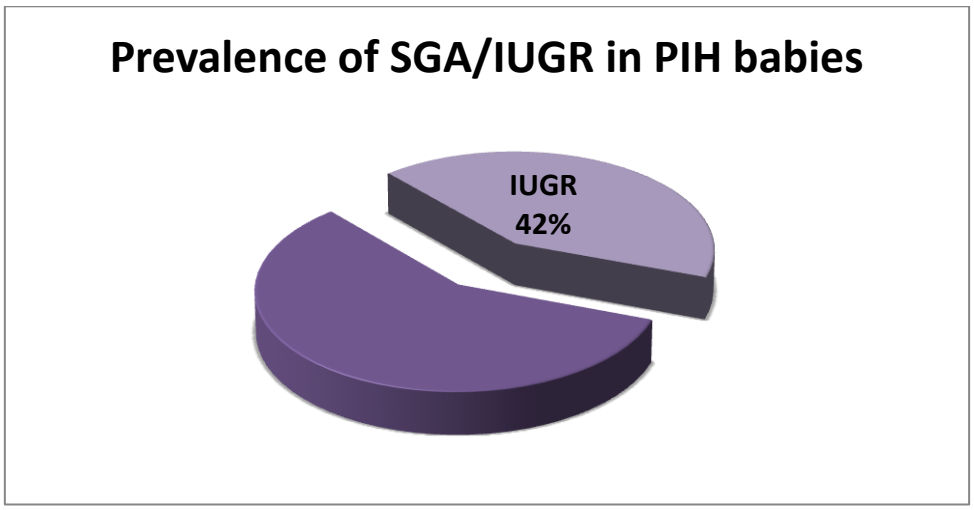

Figure 7: Percentage wise distribution of SGA/IUGR babies

5) TYPE OF IUGR

Type of IUGR	Number of babies (n=131)	Percentage	Significance of asymmetrical IUGR
Asymmetrical	93	71	p value- 0.001 (Significant)
Symmetrical	38	29	
Total	131	100	

Table 8: Frequency and Percentage wise distribution of the Type of IUGR

This shows a statistical significance of asymmetrical IUGR(p value- 0.001) in babies born to PIH mothers. This is in accordance with the uteroplacental insufficiency occurring in the later periods of gestation leading to asymmetrical IUGR.

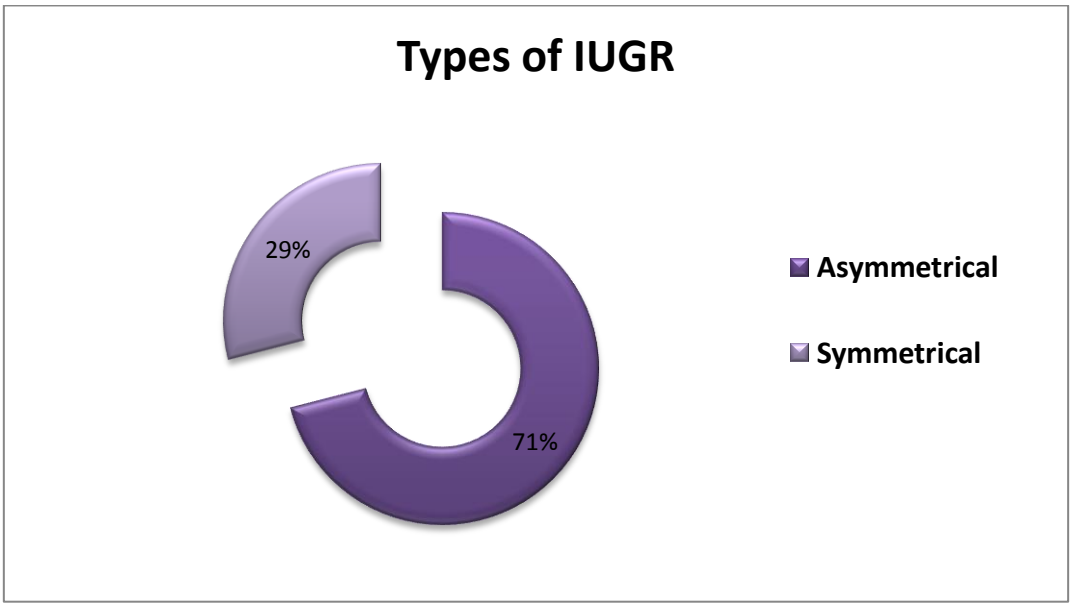

Figure 8: Percentage wise distribution of the Type of IUGR

TYPE OF IUGR	ASYMMETRICAL	SYMMETRICAL	TOTAL
PRETERM	39	38	77
TERM	54	0	54
TOTAL	93	38	131

Table 9: Gestation Age wise distribution of types of IUGR

The distribution of IUGR among preterm babies is almost equal among symmetrical and asymmetrical. But all of the term IUGR belong to the asymmetrical IUGR group.

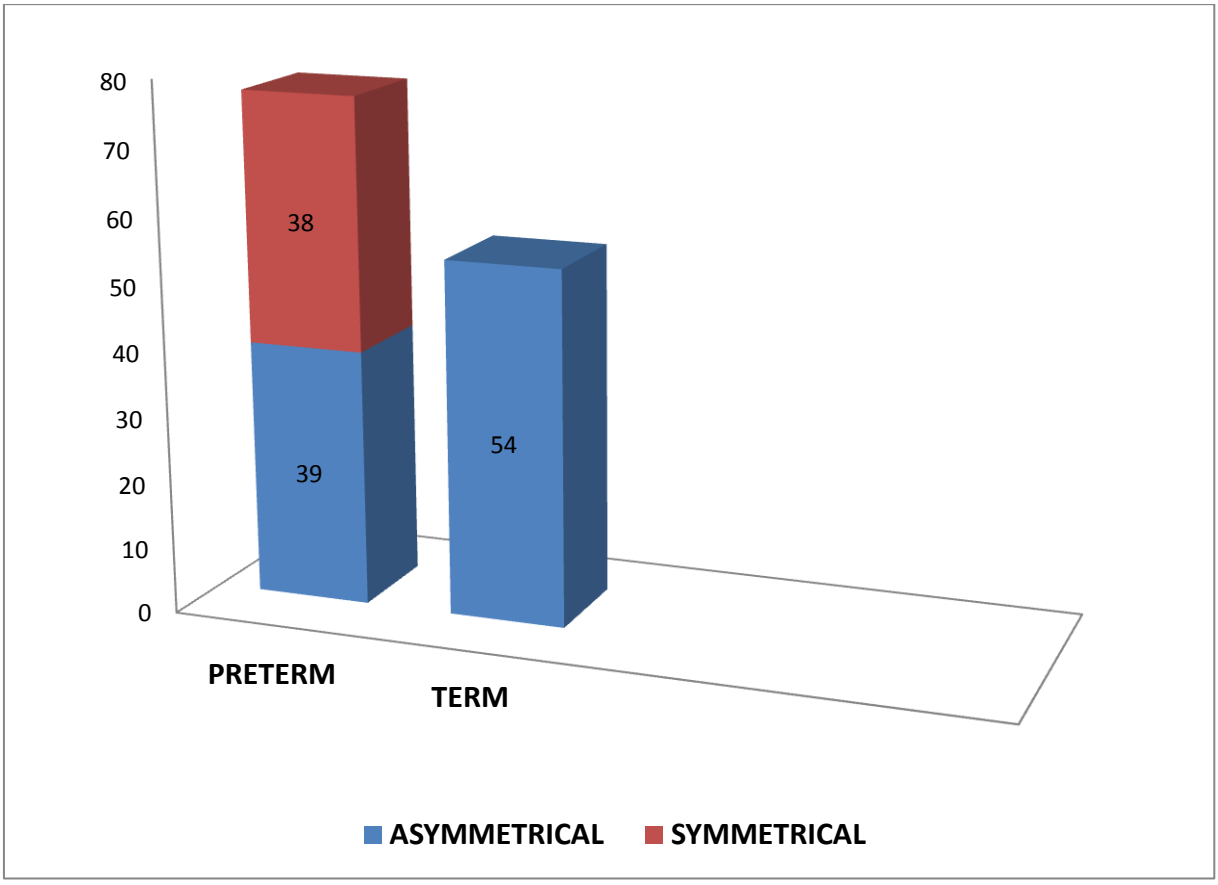

Figure 9: Gestation Age wise distribution of types of IUGR

6) LOW APGAR SCORES

APGAR SCORE<7	Number of babies	Percentage
1 MINUTE	59	18.9
5 MINUTES	14	4.5

Table 10: Frequency and Percentage wise distribution of babies with low Apgar scores

Of the total, about 18.9% have low apgar scores(<7) at the first minute. Of them, 14.4% of babies improve their apgar scores whereas 4.5% of babies continue to have low apgar scores even at the 5th minute.

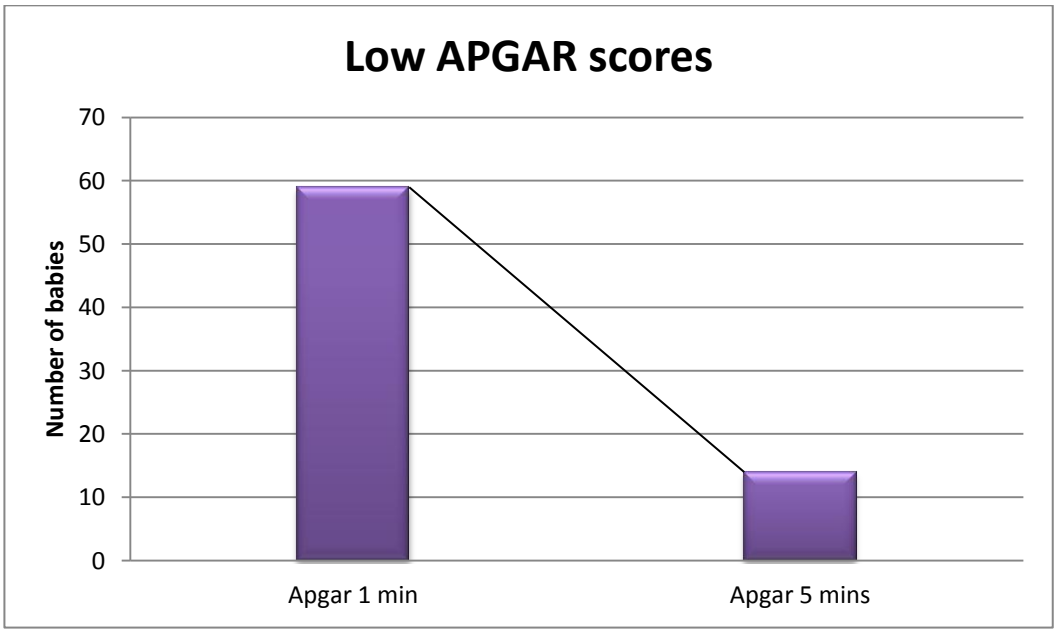

Figure 10: Distribution of babies with low Apgar scores

7) CANS SCORE

CANS score	Number of babies (n=312)	Percentage	Significance of CANS<25
>25	135	43.3	p value-0.012 (Significant)
<25	177	56.7	
Total	312	100	

Table 11: Frequency and Percentage wise distribution of babies with low CANS score

About 56.7% of all babies have Clinical Assessment of Nutritional Score <25 which implies that these proportion of babies have intrauterine malnourishment. This has a statistical significance(p value-0.012) of the total babies born to PIH mothers.

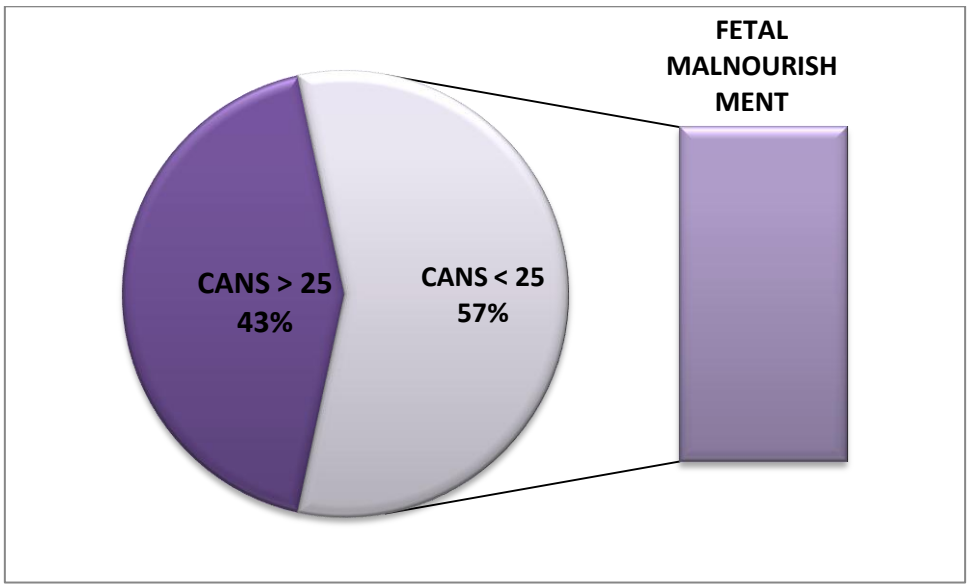

Figure 11: Percentage wise distribution of babies with low CANS score

8) NICU ADMISSION

NICU Admission	Number of babies (n=312)	Percentage
No	178	57.1
Yes	134	42.9
Total	312	100

Table 12: Frequency and Percentage wise distribution of NICU admission babies

About 42.9% of babies born to PIH mothers require admission to the Neonatal Intensive Care Unit which is found in our study.

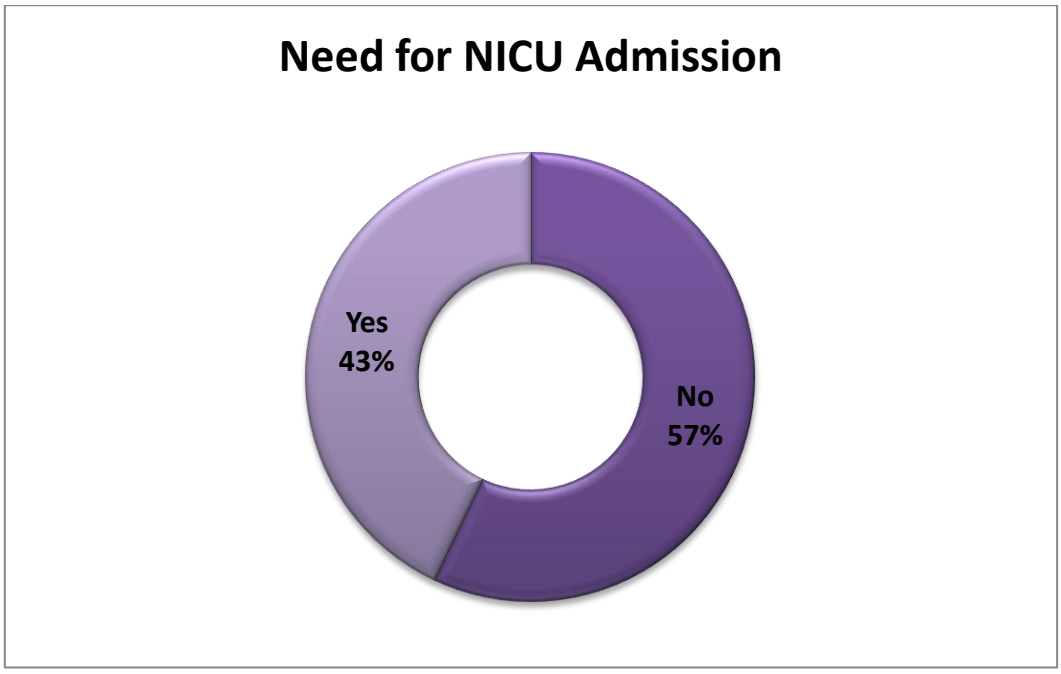

Figure 12: Percentage wise distribution of NICU admission babies

9) COMPLICATIONS

Complications	Number of babies	Percentage
Birth asphyxia	44	14.1
RDS	64	20.5
MAS	4	1.3
Sepsis	44	14.1

Table 13: Frequency and Percentage wise distribution of the complications

In our study, Respiratory Distress Syndrome(RDS) is found to be the most common complication accounting for about 20.5% of all babies. Birth asphyxia and sepsis comes next each accounting for about 14.1% of the total finally followed by Meconium Aspiration Syndrome(MAS) being the least common complication.

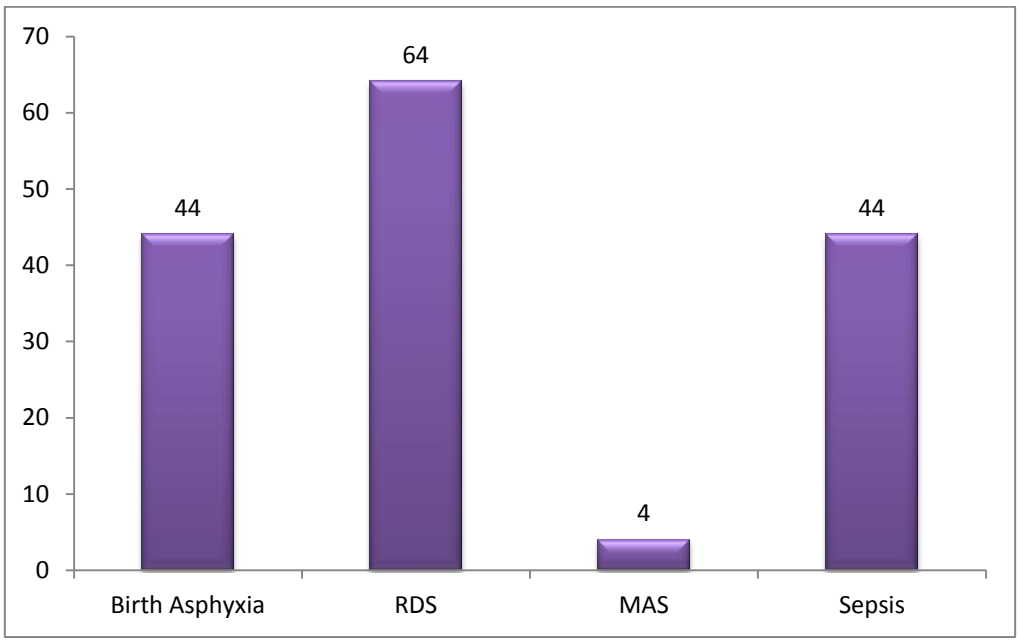

Figure 13: Frequency and distribution of the complications

10) HEMATOLOGICAL PARAMETERS

a) HEMOGLOBIN

The mean value of haemoglobin of all the babies taken into our study is 16.36 g/dl with maximum being 21.9 g/dl and minimum being 10.9g/dl.

b) TOTAL WBC COUNT

The mean value of total WBC count of all the babies taken into our study is 11000 with maximum being 23300 and minimum being 2700.

c) PLATELET COUNT

The mean value of platelet count of all the babies taken into our study is 2.11 lakhs with maximum being 6.32 lakhs and minimum being 23000.

PARAMETER STUDIED	MEAN±SD
Hemoglobin	16.36±2.03
Total WBC count	11000±4439
Platelet	2.11±0.93

Table 14: Mean values and standard deviation of the various haematological parameters

PRESENCE OF THROMBOCYTOPENIA

Platelet Count	Number of babies (n=312)	Percentage
<1.5 lakhs	88	28.2
≥1.5 lakhs	224	71.8
Total	312	100

Table 15: Frequency and Percentage wise distribution of babies based on the platelet counts

Of the total 312 babies of PIH mothers in our study, about 88 babies had platelet count < 1.5 lakhs which accounts to a total of about 28.2%.

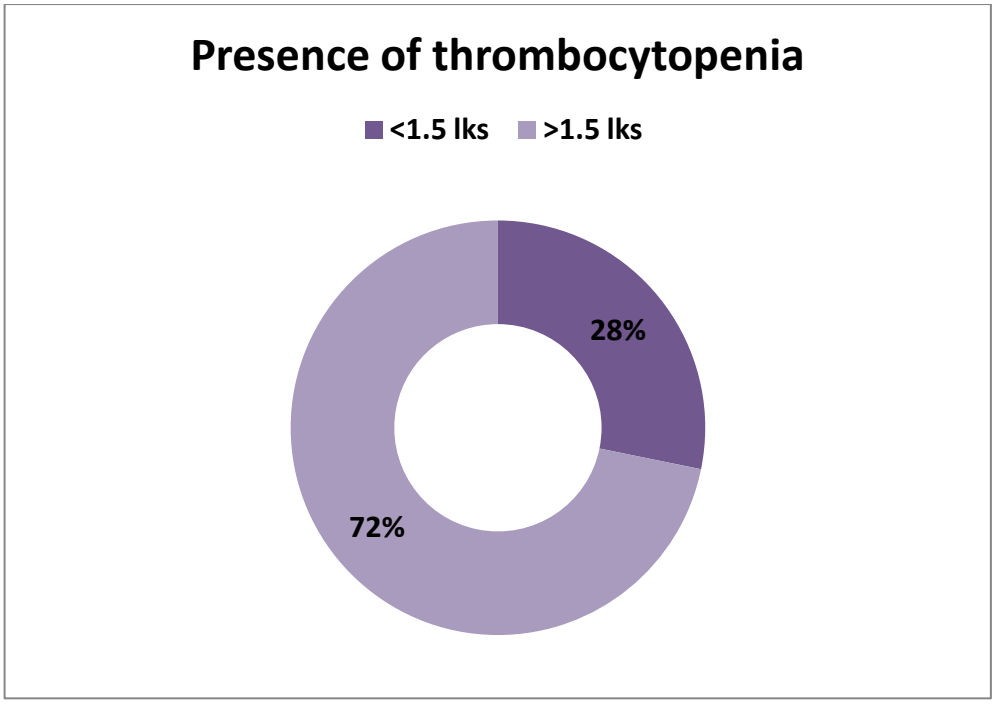

Figure 14: Percentage wise distribution of babies based on the platelet counts

DEGREES OF THROMBOCYTOPENIA	Number of babies (n=88)	Percentage
M ild(1-1.5 lakhs)	62	70.45
Moderate(50000-1 lakh)	22	25
< 50000	4	4.55

Table 16: Frequency and Percentage wise distribution of thrombocytopenic babies based on severity

Among the 88 thrombocytopenic babies born to PIH mothers, 62 babies belonged to the mild thrombocytopenia category (1-1.5 lakhs), 22 babies belonged to moderate thrombocytopenia (50000-1 lakh) and only 4 babies were severely thrombocytopenic (<50000). Thus most babies of PIH mothers had only mild thrombocytopenia.

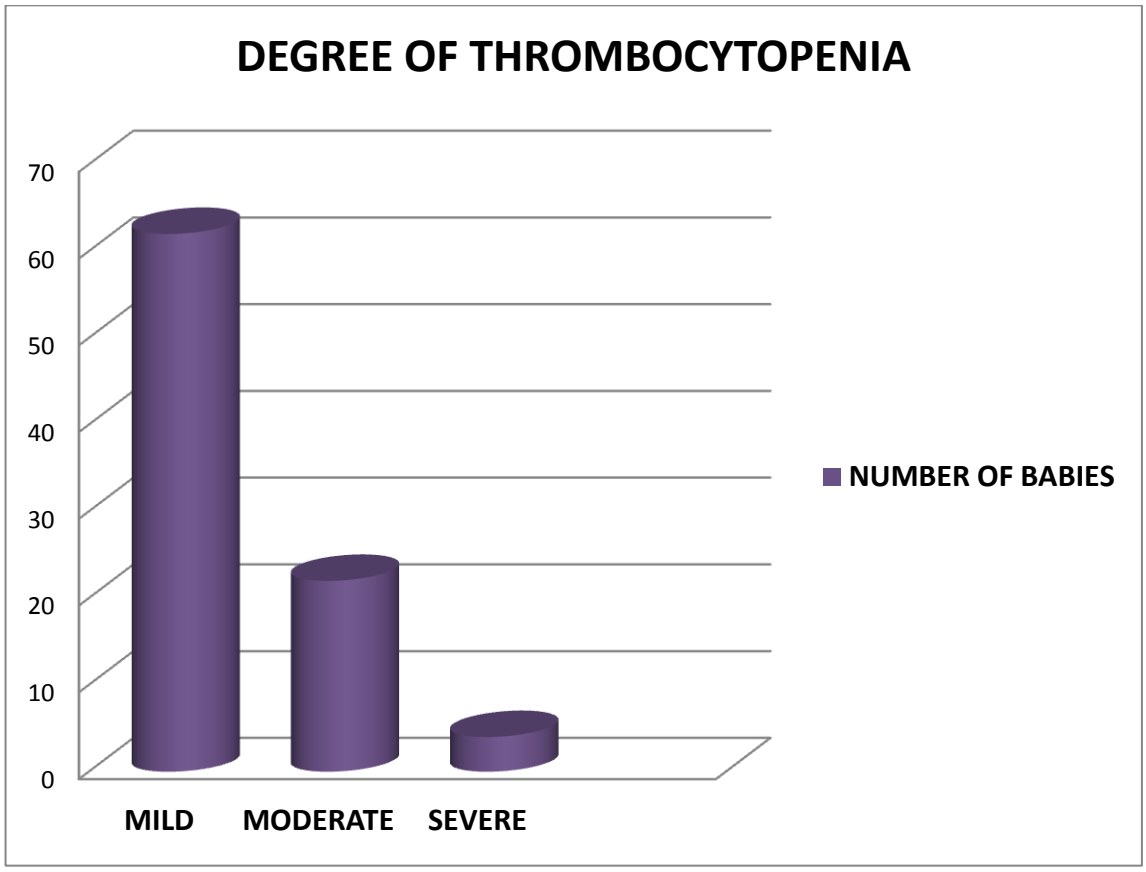

Figure 15: Distribution of thrombocytopenic babies based on severity

d) NUCLEATED RBCs

	Number of babies	Percentage
PRESENCE OF NUCLEATED RBCs	36	11.54

Table 17: Prevalence of nucleated RBCs in PIH babies

The prevalence of nucleated RBCs in the peripheral smear of the PIH babies included in our study is 11.54%

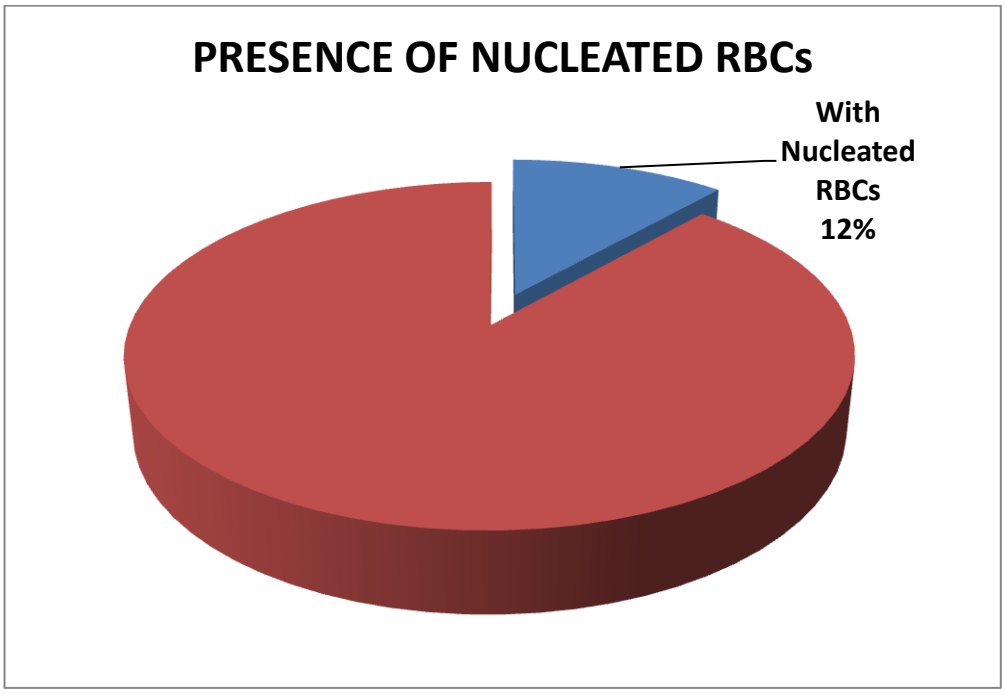

Figure 16: Prevalence of nucleated RBCs in PIH babies

11) NEONATAL DEATHS

	Number of babies	Percentage
NEONATAL DEATHS	8	2.56

Table 18: Frequency and Percentage wise distribution of deaths in PIH babies

Out of the 312 babies born to PIH mothers in our study, about 8 babies succumbed to death due to various reasons which contributes to a total of 2.56%. Out of the 8 babies, 2 babies died due to asphyxia and the other 6 babies succumbed to the complications of preterm/Low Birth Weight.

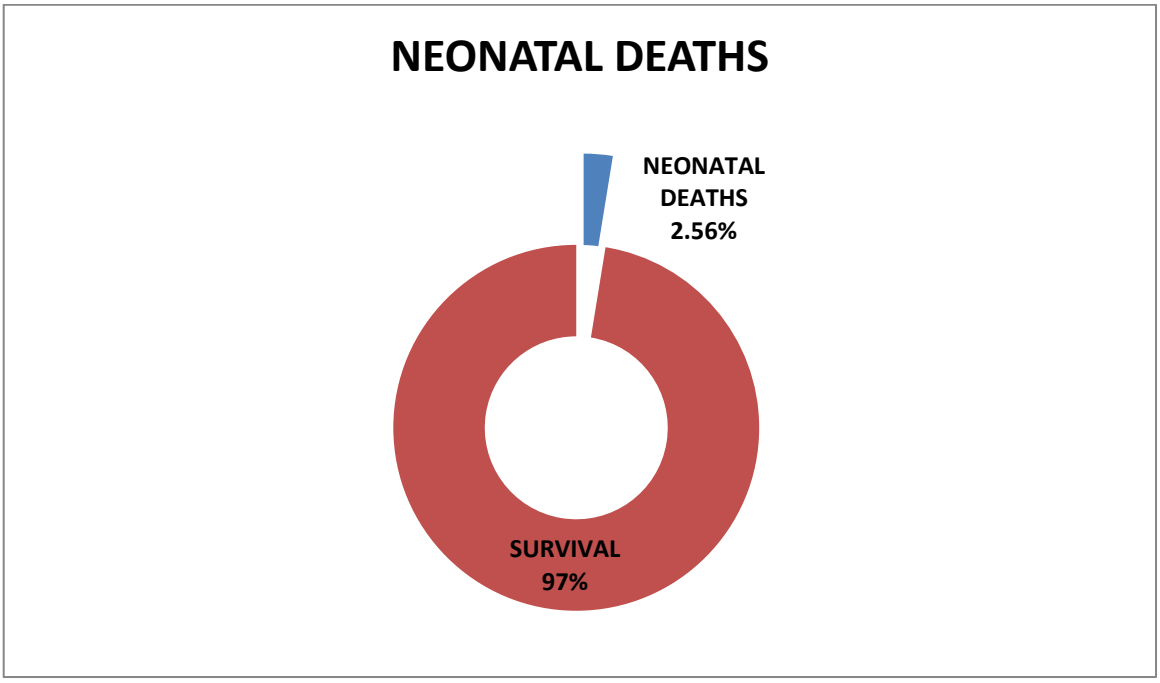

Figure 17: Percentage wise distribution of deaths in PIH babies

12) CORRELATION BETWEEN SGA/IUGR AND CANS SCORE

	Number of babies	Percentage
SGA/IUGR	131	42
ABNORMAL CANS SCORE	177	56.7

Table 19: Correlation between SGA/IUGR and CANS score

Correlation-0.743 p value-0.001

In our study, about 131 babies are identified as IUGR by Weight for Gestational Age < 10^{th} percentile whereas 177 babies are identified as fetal malnourishment by CANS score.

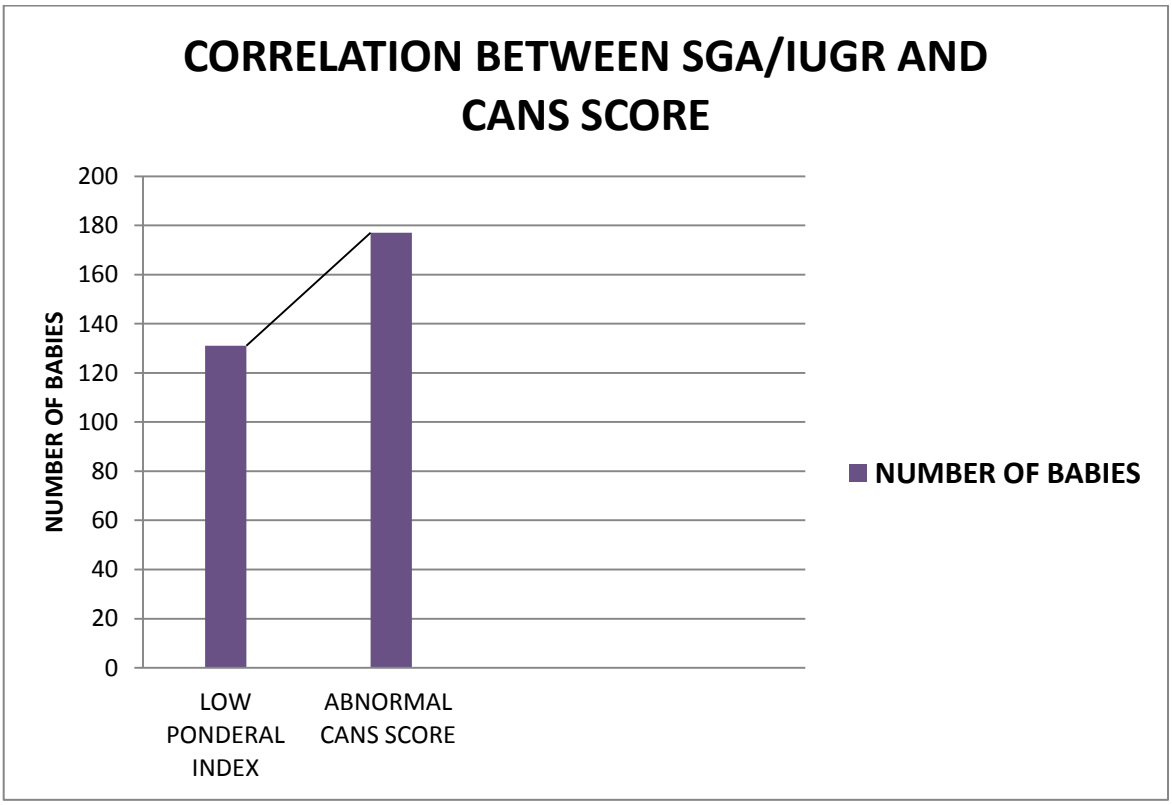

Figure 18: Correlation between SGA/IUGR and CANS score

13) CORRELATION BETWEEN IUGR AND APGAR AT 5TH MINUTE

	Number of babies	Percentage
IUGR	131	42
APGAR < 7 AT 5 MINUTE	14	4.5

Table 20: Correlation between IUGR and Apgar at 5th minute

Correlation-0.161 p value-0.004

Our study has 131 IUGR babies of which 14 babies have low APGAR scores at the 5th minute. This shows a positive statistical significance with p value- 0.004 (<0.05).

14) CORRELATION BETWEEN IUGR AND THROMBOCYTOPENIA

	Number of babies	Percentage
IUGR	131	42
THROMBOCYTOPENIA	88	28.2

Table 21: Correlation between IUGR and Thrombocytopenia

Correlation-0.153 p value-0.007

Our study proves a statistical significance between the presence of IUGR and low platelet count both due the effects of uteroplacental insufficiency.

15) CORRELATION BETWEEN LOW WBC COUNT AND SEPSIS

	Number of babies	Percentage
LOW WBC COUNT	17	5.45
SEPSIS	44	14.1

Table 22: Correlation between WBC count and Sepsis

Correlation-0.01 p value-0.864

Our study does not show a statistical significance(p value-0.864) between low WBC count and the presence of sepsis. Hence our study concluded a negative correlation between low WBC count and sepsis.

DISCUSSION

Hypertensive disorders are one of the most common obstetric complications in pregnancy. These disorders provide great challenges for obstetricians and neonatologists because they are associated with a number of adverse maternal outcomes and short and long term neonatal complications. Gestational hypertension, preeclampsia and eclampsia syndrome have important implications for the baby, suggesting that it is not a simple gestational disorder but a clinical syndrome involving important maternal and fetal vascular alterations that can persist and can cause diseases in later life[66].

The pathogenesis of preeclampsia is failure of trophoblastic invasion of the spiral arteries. The arteries will have accumulation of the fibrinoid material and the foam cells which leads to reduced blood flow and favours the interaction between the surrounding cells. Longer exposure of RBCs to oxygen metabolites and proteases produced by inflammatory cells causes RBC damage which may compromise maternal fetal exchange of oxygen and hence alters placental homeostasis and fetus development[67].

Studies have shown that hypertensive disorders of pregnancy predispose women to acute or chronic uteroplacental insufficiency, thereby having an effect on perinatal and neonatal outcome that may result in antepartum or intrapartum anoxia that may lead to fetal death, intrauterine growth retardation and/or preterm delivery[68]. Some studies have shown that there is an increased

incidence of caesarean sections in the mothers with PIH, and increased incidence of birth asphyxia, transient tachypnea of newborn(TTN), hyaline membrane disease(HMD) and neonatal sepsis in newborns of these mothers.

1) MODE OF DELIVERY

In our study, about 69.2% of babies were delivered by caesarean section which was statistically significant whereas the remaining were delivered by labor natural. This is in accordance with the study conducted by Sikha Maria Siromani et al[57] in Niloufer Hospital, Hyderabad in which about 70.67% of the PIH group mothers underwent caesarean section. Other studies like J.Nadkarni et al(35.6%), Solange Regina et al(66.7%) and Aleem Arshad et al(54.8%) also showed similar results stating an increased incidence of caesarean sections in PIH group of antenatal mothers[69,70,71].

Abdul Kareem et al[52] also stated in their study that caesarean section was the major mode of delivery in hypertensive mothers(94%). On the other hand, study conducted by Buga et al[72] in the year 1999 stated the prevalence of caesareans among the hypertensive mothers to be only 30.2%. This can be explained by the fact that at the time of the study there were limitations in diagnostic tools to assess fetal distress which is a major cause of caesarean section.

2) BIRTH WEIGHT

Our study showed a prevalence of 68.9% of low birth weight babies (<2.5kg) born to PIH mothers which is statistically significant. This is slightly higher than the other studies like the study conducted by Sikha Maria Siromani et al[57] which showed low birth weight to be 54.67% of the total PIH babies. Ravikant Patel et al[49] also showed a prevalence of 53.12% of low birth weight babies which is again lower than our study. Another study conducted by Shweta Anand et al[50] showed a prevalence of 60% low birth weight PIH babies in their study which is comparable to our study.

The weightwise distribution of the low birth weight babies showed that 89.3% of babies fall between the 1.5-2.5kg category, 9.3% babies between 1-1.5kg and only 1.4% babies have their birth weight below 1kg. This shows that in our study most LBW babies born to PIH mothers belong to the 1.5-2.5kg category.

3) GESTATIONAL AGE

Our study shows that about 60.8% of babies of PIH mothers were preterm and this proved a statistical significance in our study. This is comparable with the study conducted by Sikha Maria Siromani et al[57] which showed preterm babies of 63.01% with statistical significance. Nadkarni et al[69] showed 44.3%

preterm deliveries while Yadav et al[73] study had 28.85% and Solange Regina study[70] had 10.9%.

In our study among the 190 preterm cases, 152 babies fall under the late preterm category. Hence this represents the major subset of the preterm babies. This is in accordance with the study conducted by Carl H Backes et al[74] which states that late preterm births represent the fast growing subset of premature births from maternal preeclampsia.

4) INTRAUTERINE GROWTH RESTRICTION(IUGR)

Our study states that 42% of babies included in the study are IUGR as identified by Weight for Gestational Age < 10^{th} percentile from the Fentons intrauterine growth curves. This is in statistical significance with PIH which is proven in our study. Out of the 131 IUGR babies in our study 93 babies were asymmetrical IUGR and 38 were symmetrical IUGR. Of the 93 asymmetrical IUGR babies, 39 babies were preterm and 54 babies term. Similarly of the 38 symmetrical IUGR babies, all of them were preterm and there were no term symmetrical IUGR babies. The cumulative preterm IUGR in our study is about 58.78% of the total IUGR babies whereas the remaining constitute the term IUGR babies.

The results of our study were in accordance with the study conducted by Shweta Anand et al[50] which states that the preterm IUGR in their study group were 57.1% with 71% being asymmetrical IUGR. In contrast, preterm

asymmetrical IUGR were higher in their study group as compared to term asymmetrical IUGR.

Fetal growth retardation and PIH are thought to be initiated with improper remodelling of the uterine spiral arteries caused by inadequate trophoblast invasion in early pregnancy, leading to reduced placental and fetal perfusion and subsequent dysfunction of the maternal vascular endothelium.

5) LOW APGAR SCORES

Our study showed that the APGAR scores were < 7 in 18.9% of babies at the 1st minute whereas the scores were < 7 in only 4.5% babies at the 5th minute.

The results of our study are in accordance with the study conducted by Sulaeman A Susilo et al[51] which concluded that the APGAR score at 1 and 5th minute were 19% and 5.4% respectively. The study conducted by Abdul Kareem et al[52] also showed that the APGAR score at 5 minutes was significantly lower. But the APGAR scores at 1 and 10th minute showed no statistical difference.

In women with preeclampsia, it is often seen that there is an insufficient placental circulation. The association between abnormal placentation and preeclampsia is well known and is thought to involve in trophoblast invasion of maternal spiral arteries. Abnormal placentation results in inadequate uteroplacental blood flow that can lead to unsuccessful pregnancy outcomes. It is well known that low APGAR score most commonly results from

uteroplacental insufficiency which is a later clinical manifestation of poor placentation and placental ischaemia as caused by preeclampsia.

6) ABNORMAL CANS SCORE

Most of the classification systems for intrauterine growth retarded babies are based on observed birth weight below the 3rd or 10th percentile for gestational age estimated by use of various growth curves. However none of the classification systems identify fetal malnutrition. The clinical manifestation of fetal malnutrition depends on the timing it began during gestation. It is characterized by obvious intrauterine loss of, or failure to acquire normal amount of subcutaneous fat and muscle. Weight, length and head circumference may or may not be affected.

Our study states that 56.7% of the 312 babies included in our study have CANS score < 25 which is found to be statistically significant(p value-0.012).

7) NICU ADMISSION

Our study has identified that 42.9% of babies born to PIH mothers require NICU admission for various reasons. Most of the babies needed special nursing care in the neonatal unit either because of their preterm nature or because of low birth weight. Other co-morbid conditions like TTN, RDS, MAS and birth asphyxia were also the reasons for NICU admissions.

This is comparable to the study conducted by Sikha Maria Siromani et al[57] which concluded that 34.25% babies born to PIH group required NICU

admission which was a significant outcome of the study. Similar results were obtained in other studies like Attiya Ayaz(26.02%) and Jehan Ara(42%) study.

8) COMPLICATIONS

Various other complications such as birth asphyxia, RDS, MAS and sepsis have also been determined in this study. It states that about 14.1% suffered birth asphyxia, 20.5% Respiratory Distress Syndrome, 1.3% babies from Meconium Aspiration Syndrome and 14.1% babies suffering from sepsis.

The results of this study are comparable to the study conducted by Sikha Maria Siromani[57] which stated that 15.06% of babies in the study had birth asphyxia, 12.33% RDS, 2.73% MAS and 2.74% sepsis. Another study conducted by Nadkarni et al[69] showed similar risk of birth asphyxia(14%), and lower risk of RDS(7.3%) and sepsis(7.5%).

9) HEMATOLOGICAL PARAMETERS

This study concluded the mean haemoglobin as 16.36±2.03 g/dl, mean WBC count as 11000±4439 and the mean platelet as 2.11±0.93 lakhs. These results were comparable with the results of the study conducted by Naim Eman et al at Jawaharlal Nehru Medical College, Aligarh, Uttar Pradesh which stated the mean haemoglobin to be 16.85±3.41, mean WBC count to be 11642±6049.96 and the mean platelet count to be 164660.65±6049.96.

In an other study conducted by Abdul Karem et al[52] in Baghdad, Iraq, the results of the haematological profile of the babies of the hypertensive mothers

were mean haemoglobin- 17.27±2.29, mean WBC count-16140±5130 and the mean platelet count- 2.46±0.81. The platelet values of babies in this study were found to be comparable to our study. The study conducted by Sandhya Sivakumar et al[54] at JIPMER, Pondicherry concluded with the following results: mean haemoglobin- 17.98g/dl, mean WBC count- 14,258 and the mean platelet count- 1,94,080. The results of this study were not much correlative with our study.

The incidence of thrombocytopenia in the babies born to PIH mothers in our study is 28.2% of the total 312 babies. This is in accordance with result of the study conducted by Sandhya Sivakumar et al[54] at JIPMER, Pondicherry which stated the presence of thrombocytopenia in 22% of their cases.

Few other studies conducted by Burrows RF et al[75] and Weinstein L et al[76] recorded thrombocytopenia in 9.2% and 16% of PIH babies respectively. The study conducted by Pritchard JA et al[77] documented thrombocytopenia in only 4.2% of PIH babies. The results of these studies were lower and were not in accordance with our study. The study conducted by Sunil Kumar et al[78] in Karnataka showed thrombocytopenia in 57.3% of the PIH babies which is almost twice of that observed in our study.

In our study, thrombocytopenia was further categorized as mild(1-1.5 lakhs), moderate(50,000-1 lakh) and severe(< 50,000). About 70.45% babies fell into mild thrombocytopenia, 25% had moderate thrombocytopenia and 4.55% severe thrombocytopenia. Sandhya Sivakumar et al[54] concluded that the

platelet count were between 1-1.5 lakhs in 54.54% of thrombocytopenic babies and < 1 lakh in the remaining. Hence this was not in accordance with our study. Sunil Kumar et al[78] stated the presence of mild thrombocytopenia in 25.6% babies, moderate thrombocytopenia in 55.8% and severe thrombocytopenia in 18.6% of PIH babies. Our study had a predominance in the mild thrombocytopenia group whereas Sunil Kumar et al study stated a predominance in the moderate thrombocytopenia group.

There are studies reporting that the thrombocytopenia in the PIH babies arises from pathology at the placental level. Kleckner et al reported that abnormal placental endothelial surface caused thrombocyte destruction, and the resulting thrombocytopenia improved in a short time after delivery. But the repeat platelet counts to look for improvement in thrombocytopenia were not taken in our study.

Nucleated RBCs were present in about 12% of PIH babies in our study. The presence of nucleated RBCs in the peripheral smear of the babies born to hypertensive mothers were also demonstrated in the study conducted by Sandhya Sivakumar et al[54].

10) NEONATAL DEATHS

The total prevalence of deaths among the babies of PIH mothers in our study is 2.56%. This is comparable to the results of the study by Sikha Maria Siromani et al[57] which stated the prevalence of neonatal deaths to be 2.74%.

Study by Ravikant Patel et al[49] showed a neonatal death prevalence of 1.56% in babies of hypertensive mothers which is found to be lower than our study. Bangal et al in their study at a rural tertiary level health care referral centre in Loni, Maharashtra concluded their result with a neonatal death prevalence of 5% in PIH babies which when compared with our study is much higher.

LIMITATIONS OF THE STUDY

- The babies of hypertensive mothers were not categorized separately as babies of gestational hypertension, preeclampsia and eclampsia and studied in separate groups. Babies of hypertensive mothers included in our study were studied together. Hence stratified analysis of the babies was not done.

- Follow up of the babies were not done and hence the long term complications could not be looked upon.

- The haematological profile of the mothers were not recorded and hence the correlation between neonates and mothers haematological parameters could not be studied.

CONCLUSION

Pregnancy Induced Hypertension is a maternal pathology involving placental modification which is associated with foetal complications. Foetal morbidity and mortality are serious concerns in preeclampsia and are attributable to poor management. Since babies born to hypertensive mothers are prone to develop several complications, close monitoring of these babies should be undertaken in an attempt to provide these babies with decreased morbidity and improved growth development and survival. The causes for perinatal mortality in this group of babies are mainly prematurity and low birth weight. Hence proper antenatal care must be given to all pregnant women to prevent and screen for preeclampsia. Public health awareness, education of the primary health care workers and improvement of socio-economic circumstances can help to improve the neonatal prognosis.

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PROFORMA

1. **Name:**
2. **Sex:**
3. **Ip/Op No:**
4. **Date & time of birth:**
5. **Mode of delivery: LN/LSCS (Indication)**
6. **Complications of labour(if any):**
7. **Apgar Score: 1”-**

   5”-

   NICU Admission: Yes/No

   Reason:
8. **Maternal details:**

   Parity

   Booked/Immunised

   Proteinuria

   Recurrence of PIH

   Antihypertensive Medications

   Antenatal Steroids

   Antenatal USG(after 20wks)

9. **Baby Details:**

   Birth weight

Length

Gestational Age

Ponderal Index

CANS score

New Ballard Score

Neuromuscular maturity

Physical maturity

Head circumference

Chest circumference

Birth asphyxia

Respiratory distress

Meconium aspiration

Sepsis

10. Hematological parameters (only in babies admitted in NICU)

Hb

WBC

Platelet

Peripheral Smear

11. Outcome: Discharged/ Death (Cause of death)

Sl.No	Name	Mode of delivery	Birth weight(kg	Gestational Age	IUGR	Type of IUGR	Apgar<7(1 min)	Apgar<7(5 min)	CANS score	NICU Admission	Reason	Birth Asphyxia	RDS	MAS	Sepsis	Neonatal D	Hb(g/dl)	TC	Platelet(lak	Nucleated RBCs
1	B/o Vanitha	LSCS	2.2	36 wks					<25	Yes	Resp distress						16.7	8400	1.83	
2	B/o Panchavarnam	LSCS	1.8	35 wks	Yes	Asymmetrical			<25	Yes	Preterm/LBW		Yes				15.4	5100	1.02	Yes
3	B/o Nithya	LSCS	2.8	39 wks						No							17.1	14800	2.52	
4	B/o Shanthi	LSCS	1.6	36 wks	Yes	Asymmetrical			<25	Yes	Resp distress						15.3	11300	1.61	
5	B/o Nadhiya	LN	2.3	39 wks	Yes	Asymmetrical			<25	No							14.1	9200	3.23	
6	B/o Shanmuga Priya	LN	2.6	38 wks						No							15.6	12500	2.83	
7	B/o Sathya Priya	LSCS	3	39 wks						Yes	MAS	Yes		Yes			15.9	7100	1.9	
8	B/o Fathima Banu	LSCS	1.2	34 wks	Yes	Asymmetrical			<25	Yes	Preterm/IUGR	Yes	Yes				13.3	15700	1.52	
9	B/o Radhika	LSCS	1.1	30 wks	Yes	Symmetrical			<25	Yes	Preterm/LBW		Yes		Yes	Yes	19.2	6200	0.96	Yes
10	B/o Thilagavani	LN	2.8	39 wks						No							18.8	12600	2.36	
11	B/o Sathya	LSCS	2.9	39 wks						No							16.4	13300	2.1	
12	B/o Banumathi	LSCS	1.8	36 wks	Yes	Asymmetrical	Yes	Yes	<25	Yes	Asphyxia	Yes					12.9	23300	2.2	
13	B/o Suganthi	LSCS	3.3	39 wks						No							15.5	14200	3.1	
14	B/o Alagulakshmi	LSCS	1.3	34 wks	Yes	Symmetrical			<25	Yes	Preterm/IUGR		Yes		Yes		17.4	5500	1.01	Yes
15	B/o Kamala	LSCS	2.1	36 wks			Yes		<25	Yes	Asphyxia	Yes					14.6	16400	1.85	
16	B/o Alagurani	LN	1.4	33 wks	Yes	Symmetrical			<25	Yes	Preterm/LBW		Yes				17.1	20400	1.36	Yes
17	B/o Muthulakshmi	LN	2.8	38 wks						No							16.5	14400	2.2	
18	B/o Lakshmi	LN	2.9	39 wks						No							15.9	11300	2.35	
19	B/o Lingeshwari	LSCS	1.9	35 wks	Yes	Asymmetrical			<25	Yes	Preterm/LBW				Yes		12.9	18100	1.5	
20	B/o Nagalakshmi	LSCS	2.2	35 wks			Yes	Yes		Yes	Asphyxia	Yes					16.6	6300	0.54	
21	B/o Jothi	LSCS	2.5	36 wks			Yes			Yes	Asphyxia	Yes					21.2	14400	0.86	
22	B/o Pownmari	LSCS	2.1	38 wks	Yes	Asymmetrical			<25	No							15.2	10500	2.54	
23	B/o Anbarasi	LN	2.4	36 wks						Yes	Resp distress						14.6	16200	1.96	
24	B/o Nagajothi	LN	2.6	39 wks					<25	No							19.1	17100	3.2	
25	B/o Meena	LSCS	2	36 wks	Yes	Asymmetrical			<25	Yes	Resp distress						13.2	19600	6.32	
26	B/o Nithya	LSCS	2.3	39 wks	Yes	Asymmetrical			<25	No							15.2	13500	2.35	
27	B/o Seethalakshmi	LSCS	1.9	34 wks					<25	Yes	Preterm/LBW		Yes				20.2	5600	1.36	
28	B/o Kanakadevi	LN	1.6	34 wks	Yes	Asymmetrical	Yes		<25	Yes	Preterm/LBW	Yes	Yes		Yes		17.4	8100	0.66	Yes
29	B/o Jelin	LSCS	3.1	39 wks						No							16	15400	2.1	
30	B/o Rajalakshmi	LN	2.2	37 wks	Yes	Asymmetrical	Yes		<25	Yes	MAS			Yes			17.4	20500	4.84	
31	B/o Pattamal	LSCS	2.4	36 wks						Yes	Resp distress						16.6	5100	1.74	
32	B/o Hemalatha	LSCS	2.7	38 wks						No							13.5	15400	1.23	
33	B/o Indhurani	LSCS	2.3	36 wks						Yes	Resp distress						16.1	12500	2.4	
34	B/o Thangamani	LN	1.2	32 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/LBW	Yes	Yes		Yes		14.2	4900	1.23	Yes
35	B/o Muthumeena	LSCS	3	39 wks						No							16.6	14300	2.6	
36	B/o Pothumani	LSCS	3.3	40 wks						No							17.1	16700	2.35	
37	B/o Rani	LSCS	2.5	36 wks			Yes	Yes		Yes	Asphyxia	Yes					16.1	19600	1.96	
38	B/o Sundari	LSCS	1.9	35 wks	Yes	Asymmetrical			<25	Yes	Preterm/LBW						19.5	6100	1.36	
39	B/o Kanaka	LN	2.2	36 wks					<25	Yes	Resp distress						15.4	14500	2.25	
40	B/o Annapriya	LN	2.3	39 wks	Yes	Asymmetrical			<25	No							16.1	15100	2.89	
41	B/o Divya	LN	1.7	34 wks	Yes	Symmetrical			<25	Yes	Preterm/LBW		Yes				20.2	7600	1.4	Yes
42	B/o Muthulakshmi	LSCS	3.1	39 wks						Yes	MAS			Yes			19.3	20300	3.64	
43	B/o Mithrapriya	LSCS	2.4	36 wks						No							17.7	11300	3.1	
44	B/o Pandilakshmi	LSCS	2.4	36 wks			Yes			Yes	Resp distress						13	14000	2.76	
45	B/o Menaka	LSCS	2.7	38 wks						No							15.6	16100	3.3	
46	B/o Nithya	LSCS	1.9	33 wks	Yes	Asymmetrical			<25	Yes	Preterm/LBW		Yes		Yes	Yes	13.2	16400	1.43	
47	B/o Mahalakshmi	LN	1.8	34 wks	Yes	Symmetrical			<25	Yes	Preterm/LBW	Yes	Yes				14.9	19400	1.32	Yes
48	B/o Illakiya	LN	2.6	40 wks					<25	No							15.5	15500	2.36	
49	B/o Velmani	LSCS	1.5	33 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/IUGR		Yes		Yes		16.3	5400	1.44	
50	B/o Aparna	LSCS	2.3	39 wks	Yes	Asymmetrical			<25	No							18.2	12800	3.65	
51	B/o Vimala	LSCS	2.3	36 wks						No							17.6	16500	3.3	
52	B/o Veeralakshmi	LN	1.9	35 wks	Yes	Asymmetrical			<25	Yes	Preterm/LBW						12.3	8100	1.92	
53	B/o Pandeeswari	LN	2.8	40 wks						No							13.6	14400	2.56	
54	B/o Nagajothi	LSCS	2.6	39 wks						No							15.4	8100	2.12	
55	B/o Kalaivani	LSCS	1.9	36 wks	Yes	Asymmetrical	Yes		<25	Yes	LBW						13.2	5300	1.69	
56	B/o Vijayalakshmi	LSCS	2.3	39 wks	Yes	Asymmetrical			<25	No							15.6	13500	3.6	
57	B/o Pavithra	LSCS	2.4	36 wks						No							18.1	20100	4.75	
58	B/o Sangeetha	LSCS	2.1	35 wks					<25	Yes	Preterm						20.2	8500	2.65	
59	B/o Chithra	LSCS	3.2	40 wks						No							16.5	12300	1.74	
60	B/o Vennila	LN	0.9	31 wks	Yes	Symmetrical	Yes	Yes	<25	Yes	Preterm/LBW	Yes	Yes		Yes	Yes	17.2	9600	0.69	Yes
61	B/o Umarani	LSCS	2.8	39 wks						No							14.5	16300	1.43	
62	B/o Priya	LSCS	2.5	36 wks						No							15.1	8100	1.58	
63	B/o Gayathri	LSCS	2.6	38 wks						No							16.6	12600	2.66	
64	B/o Muthulakshmi	LSCS	1.4	33 wks	Yes	Asymmetrical			<25	Yes	Preterm/LBW		Yes		Yes		19.9	18800	1.32	Yes
65	B/o Archana	LSCS	3.7	41 wks						No							17.7	11300	2.73	
66	B/o Kalaiselvi	LSCS	2.6	39 wks						No							13.4	10500	3.34	
67	B/o Anitha	LSCS	2.4	36 wks						No							16.9	7800	2.68	
68	B/o Meena	LN	1.3	32 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/LBW	Yes	Yes				15.4	17400	1.2	Yes
69	B/o Bharathi	LN	2.2	36 wks					<25	Yes	Resp distress						15.5	12300	1.55	
70	B/o Nandhini	LSCS	3	39 wks						No							15.1	13900	2.59	
71	B/o Hemalatha	LSCS	1.8	35 wks	Yes	Asymmetrical			<25	Yes	Preterm/LBW	Yes					16.3	13600	1.33	
72	B/o Sivalakshmi	LSCS	1.8	36 wks	Yes	Asymmetrical	Yes		<25	Yes	Resp distress						19.1	11900	1.91	
73	B/o Karimozhi	LN	2.4	40 wks	Yes	Asymmetrical			<25	No							17.7	9800	2.98	
74	B/o Priya	LN	2.5	39 wks					<25	No							16.4	10300	2.63	
75	B/o Rampriya	LN	1.7	35 wks	Yes	Asymmetrical			<25	Yes	Preterm/LBW				Yes		17.1	12400	1.74	
76	B/o Vennila	LSCS	2.3	36 wks						No							17.9	13800	2.93	
77	B/o Suganthi	LSCS	2.3	39 wks	Yes	Asymmetrical			<25	No							15.2	14500	2.55	
78	B/o Naveena	LSCS	2.8	40 wks						No							13.3	9100	2.19	
79	B/o Geetha	LSCS	1.9	34 wks						Yes	Preterm/LBW		Yes				19.9	8500	1.58	
80	B/o Kalpana	LSCS	2.4	36 wks						No							12.5	15200	1.35	
81	B/o Chinnakutti	LN	1.2	33 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/IUGR	Yes	Yes		Yes		18.5	6300	2.86	Yes
82	B/o Karthika	LSCS	1.7	35 wks	Yes	Asymmetrical	Yes		<25	Yes	Preterm/LBW				Yes		17.7	19400	1.47	
83	B/o Pandeeswari	LSCS	3	39 wks						No							16.3	11500	4.15	
84	B/o Sobiya	LSCS	2.5	39 wks					<25	No							16.6	12400	2.46	
85	B/o Rubeena Banu	LN	1.5	34 wks	Yes	Symmetrical			<25	Yes	Preterm/LBW	Yes	Yes				15.9	9600	1.95	
86	B/o Thangeswari	LSCS	2	36 wks					<25	Yes	Resp distress						16.9	15500	2.4	
87	B/o Pitchaimani	LSCS	2.4	40 wks	Yes	Asymmetrical			<25	No							16.2	11300	3.35	
88	B/o Anitha	LSCS	3.3	41 wks					<25	No							18.6	16400	1.39	
89	B/o Eswari	LSCS	2.7	39 wks					<25	No							15.8	18700	2.69	
90	B/o Angaleaswari	LN	1.4	33 wks	Yes	Symmetrical	Yes	Yes	<25	Yes	Preterm/LBW	Yes	Yes				18.6	5200	2.21	Yes
91	B/o Saraswathy	LN	2.8	40 wks						No							17.4	14400	4.7	
92	B/o Selvi	LN	1.8	34 wks					<25	Yes	Preterm/LBW						15.2	12500	1.55	
93	B/o Balanathiya	LN	2.3	39 wks	Yes	Asymmetrical			<25	No							16.3	12600	2.85	
94	B/o Anitha	LN	3	40 wks						No							17.4	17700	2.35	
95	B/o Nandhini	LSCS	2.4	36 wks						No							15.3	12600	3.15	
96	B/o Meena	LN	1.7	36 wks	Yes	Asymmetrical	Yes		<25	Yes	Resp distress						13.3	15900	2.01	
97	B/o Selvi	LSCS	2.8	40 wks						No							16.6	15800	2.2	
98	B/o Karthiga	LSCS	1.7	35 wks	Yes	Asymmetrical			<25	Yes	Preterm/LBW				Yes	Yes	17.5	14700	1.54	
99	B/o Muthuselvi	LSCS	1.8	36 wks	Yes	Asymmetrical			<25	No							15.6	12600	2.65	
100	B/o Abirami	LN	2	36 wks					<25	Yes							16.5	15500	1.1	Yes
101	B/o Anandhi	LSCS	2.4	41 wks	Yes	Asymmetrical			<25	No							15.5	12300	2.12	
102	B/o Gayathri	LSCS	2.2	40 wks	Yes	Asymmetrical			<25	No							13.9	14800	1.89	
103	B/o Jeyanthi	LSCS	2.9	41 wks						Yes							16.2	12600	2.53	
104	B/o Sharmila	LSCS	1.8	34 wks			Yes		<25	Yes	Preterm/LBW	Yes	Yes				18.7	9500	1.84	
105	B/o Gayathri	LN	2.2	36 wks					<25	No							17.1	13500	2.57	
106	B/o Durgeswari	LSCS	2.4	36 wks						No							16.6	13300	3.63	
107	B/o Malar	LSCS	2	35 wks						Yes	Preterm/LBW		Yes				19.2	6600	1.26	Yes
108	B/o Rajalakshmi	LSCS	2.1	40 wks	Yes	Asymmetrical			<25	No							15.4	14500	2.47	
109	B/o Ponkothai	LN	1.5	33 wks	Yes	Symmetrical	Yes	Yes	<25	Yes	Preterm/LBW	Yes	Yes		Yes		17.6	6700	1.93	Yes
110	B/o Sumithra	LSCS	2.5	39 wks					<25	No							17.1	8600	3.65	
111	B/o Amutha	LSCS	2.4	40 wks	Yes	Asymmetrical			<25	No							16.6	9900	2.31	
112	B/o Murugeswari	LN	2.7	40 wks						No							18.5	17500	2.78	
113	B/o Nithya	LSCS	2	35 wks			Yes			Yes	Preterm/LBW						19.1	13900	1.25	
114	B/o Kasthuri	LSCS	2.5	36 wks						No							16.3	13600	2.42	
115	B/o Rajathi	LSCS	1.8	36 wks	Yes	Symmetrical			<25	Yes	Resp distress		Yes		Yes		13.5	12600	1.02	Yes
116	B/o Dhanam	LSCS	3.1	40 wks						No							14.3	10200	2.61	
117	B/o Devi	LSCS	2.1	36 wks					<25	Yes	Resp distress		Yes				15.6	17700	1.53	
118	B/o Nandhini	LSCS	2.4	39 wks	Yes	Asymmetrical			<25	No							15.2	12300	2.32	
119	B/o Gayathri	LN	2.4	36 wks						No							16.6	16700	2.7	
120	B/o Eswari	LN	1.4	33 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/LBW	Yes	Yes		Yes	Yes	14.3	12600	1.43	Yes
121	B/o Amutha	LSCS	1.9	35 wks	Yes	Symmetrical			<25	Yes	Preterm/LBW				Yes		21.2	6100	2.21	
122	B/o Gayathri	LSCS	1.8	36 wks	Yes	Asymmetrical			<25	No							14.6	11100	4.13	
123	B/o Saranya	LSCS	2.2	36 wks			Yes		<25	Yes	Asphyxia	Yes					16.3	9600	3.24	
124	B/o Rajeswari	LSCS	2.4	39 wks	Yes	Asymmetrical			<25	No							15.9	12300	2.1	
125	B/o Kalaivani	LSCS	2.2	40 wks	Yes	Asymmetrical			<25	No							16	8900	3.45	
126	B/o Manjula	LSCS	2	35 wks						Yes	Preterm/LBW				Yes		10.9	5400	1.2	
127	B/o Maheswari	LSCS	2.6	36 wks						No							14.3	18700	2.33	
128	B/o Sarabunisha	LN	1.5	33 wks	Yes	Symmetrical	Yes	Yes	<25	Yes	Preterm/LBW		Yes		Yes		18.2	8200	1.74	
129	B/o Divya	LSCS	3.3	40 wks						No							17.1	14600	2.2	
130	B/o Narmadha	LSCS	2.3	39 wks	Yes	Asymmetrical			<25	No							17.7	12300	3.67	
131	B/o Balasudha	LSCS	1.6	34 wks	Yes	Asymmetrical	Yes		<25	Yes	Preterm/LBW	Yes	Yes				19.5	4200	1.55	
132	B/o Ramyadevi	LSCS	2.5	36 wks						No							16.6	12100	2	
133	B/o Ponnuthai	LSCS	2.4	36 wks						No							12.3	15400	1.23	
134	B/o Jerima Banu	LN	2	40 wks	Yes	Asymmetrical			<25	No							15.6	13200	2.45	
135	B/o Kaleeswari	LSCS	2.4	36 wks						No							17.1	8900	2.89	
136	B/o Parimala	LSCS	2.3	39 wks	Yes	Asymmetrical	Yes		<25	Yes	MAS	Yes		Yes			13.7	6100	2.2	
137	B/o Nandhini	LSCS	2.3	39 wks	Yes	Asymmetrical			<25	No							16.5	9600	4.15	
138	B/o Palaniyammal	LSCS	2.5	36 wks						No							17.2	12400	2.13	
139	B/o Suganya	LSCS	1.7	35 wks	Yes	Asymmetrical			<25	Yes	Preterm/LBW	Yes	Yes		Yes		18.6	6700	1.97	
140	B/o Vennila	LSCS	2.9	40 wks						No							19	9400	1.32	
141	B/o Mahalakshmi	LSCS	1.6	34 wks	Yes	Symmetrical			<25	Yes	Preterm/LBW		Yes				16.9	5600	1.1	Yes
142	B/o Angel Anitha	LN	2.1	36 wks					<25	Yes	Preterm/LBW						17.7	3100	1.81	
143	B/o Vasundhra devi	LSCS	2.7	39 wks						No							16.5	6200	3.2	
144	B/o Selvapriya	LSCS	2.2	35 wks						Yes	Preterm/LBW						21.2	15100	1.67	
145	B/o Rajeswari	LSCS	3.2	40 wks						No							17.6	9100	2.25	
146	B/o Jemma	LN	1.5	36 wks	Yes	Asymmetrical	Yes	Yes	<25	Yes	Preterm/IUGR				Yes		16.3	4500	0.66	
147	B/o Suganya	LSCS	2.4	36 wks						No							14.2	8100	2.23	
148	B/o Surya	LSCS	1.8	35 wks	Yes	Symmetrical			<25	Yes	Preterm/LBW						12.1	13300	0.92	
149	B/o Chithra	LSCS	2.1	40 wks	Yes	Asymmetrical			<25	No							16.6	15100	1.72	
150	B/o Murugeswari	LSCS	2.2	36 wks					<25	Yes	Resp distress		Yes				16.3	20200	3.32	
151	B/o Eswari	LSCS	2.5	36 wks						No							17.7	14600	2.8	
152	B/o Vinitha	LSCS	2.4	40 wks	Yes	Asymmetrical			<25	No							14.2	8400	2.13	
153	B/o Priyanka	LN	2.9	39 wks						No							16.1	12300	2.33	
154	B/o Alagusundari	LN	2.7	38 wks						No							18.3	8100	4.15	
155	B/o Keerthana	LN	1.9	34 wks			Yes		<25	Yes	Preterm/LBW	Yes	Yes		Yes		18.6	9600	1.23	

Sl.No	Name	Mode of delivery	Birth weight(kg	Gestational Age	IUGR	Type of IUGR	Apgar<7(1 min)	Apgar<7(5 min)	CANS score	NICU Admission	Reason	Birth Asphyxia	RDS	MAS	Sepsis	Neonatal D	Hb(g/dl)	TC	Platelet(lak	Nucleated RBCs
156	B/o Karunya	LSCS	2.8	39 wks						No							14.2	7900	3.1	
157	B/o Nirmala	LSCS	1.8	36 wks	Yes	Symmetrical			<25	Yes	Resp distress		Yes				12.6	10500	2.32	
158	B/o Anitha	LSCS	2.2	35 wks						Yes	Preterm/LBW						11.3	5400	1.7	
159	B/o Silambarasi	LSCS	2	36 wks			Yes		<25	Yes	Asphyxia	Yes				Yes	11.6	20600	1.42	
160	B/o Vajitha	LSCS	1.7	36 wks	Yes	Asymmetrical			<25	Yes	Preterm/LBW	Yes	Yes				17.9	4200	0.96	Yes
161	B/o Suganya	LSCS	3.1	40 wks						No							16.5	12200	2.89	
162	B/o Nithya	LSCS	2.8	36 wks						No							14.3	13400	0.63	
163	B/o Iswarya	LSCS	1.1	38 wks	Yes	Asymmetrical			<25	Yes	IUGR	Yes					19.5	5900	1.44	
164	B/o Viji	LSCS	0.9	34 wks	Yes	Asymmetrical			<25	Yes	Preterm/IUGR		Yes		Yes		15.5	5500	1.2	Yes
165	B/o Backiyalakshmi	LSCS	2	38 wks	Yes	Asymmetrical	Yes		<25	Yes	Asphyxia	Yes			Yes		18.3	21300	4.56	
166	B/o Neetha	LSCS	2.5	36 wks						No							14.5	10500	2.23	
167	B/o Rajammal	LSCS	2.9	40 wks						No							12.9	9200	3.1	
168	B/o Arockiya Vidhya	LN	2.4	36 wks						No							14.9	8500	2.5	
169	B/o Sangeetha	LSCS	3	40 wks						No							16.1	6400	3.23	
170	B/o Perumayee	LN	1.1	35 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/IUGR				Yes		19.2	12900	0.83	
171	B/o Pavithra	LSCS	2.3	36 wks						No							18.3	8600	2.25	
172	B/o Karthika	LSCS	1.8	36 wks	Yes	Asymmetrical			<25	Yes	Resp distress		Yes		Yes		12.2	14500	1.74	
173	B/o Surya	LSCS	2.3	40 wks	Yes	Asymmetrical			<25	No							15.6	9100	3.3	
174	B/o Srivijayam	LN	2.2	35 wks			Yes			Yes	Preterm/LBW						16.4	8200	1.69	
175	B/o Kowsalya	LN	1.7	35 wks	Yes	Asymmetrical			<25	Yes	Preterm/LBW	Yes	Yes				16.9	9600	2.46	
176	B/o Sindhya	LN	3.3	40 wks						No							17.8	13400	1.13	
177	B/o Mahalakshmi	LSCS	3	40 wks						No							18.1	15500	2.25	
178	B/o Divya	LSCS	2.5	36 wks						No							12.6	7200	2.79	
179	B/o Divyapriya	LSCS	1.7	35 wks	Yes	Asymmetrical			<25	Yes	Preterm/LBW						15.5	2700	1.84	
180	B/o Nandhini	LSCS	2.4	36 wks						No							16.7	8900	2.2	
181	B/o Stella	LN	2.4	35 wks						Yes	Preterm/LBW						12.3	13300	1.12	
182	B/o Jeyalakshmi	LSCS	2.4	39 wks	Yes	Asymmetrical			<25	No							14.8	5400	2.56	
183	B/o Mariammal	LSCS	2.5	36 wks						No							16.6	9400	2.89	
184	B/o Aarthi	LSCS	2	39 wks	Yes	Asymmetrical			<25	No							16.1	7100	3.6	
185	B/o Pushpa	LN	2.2	36 wks					<25	No							14.2	13200	2.57	
186	B/o Agnesmercy	LN	2.4	38 wks	Yes	Asymmetrical			<25	No							17.3	11300	1.89	
187	B/o Prema	LSCS	2.5	36 wks						No							16.5	14700	0.96	
188	B/o Viji	LN	1.5	34 wks	Yes	Symmetrical	Yes	Yes	<25	Yes	Preterm/IUGR				Yes		20.1	5100	0.44	Yes
189	B/o Kaleeswari	LSCS	1.9	34 wks			Yes		<25	Yes	Preterm/LBW	Yes					19.2	6700	1.84	Yes
190	B/o Renganayaki	LSCS	2.2	38 wks	Yes	Asymmetrical			<25	No							17.6	12300	1.4	
191	B/o Krishnakumari	LN	2.2	36 wks					<25	No							12.3	14100	2.23	
192	B/o Backiyalakshmi	LSCS	2.8	39 wks						No							15.9	9600	1.25	
193	B/o Nagajothi	LN	1.8	36 wks	Yes	Asymmetrical			<25	Yes	Resp distress		Yes				16.1	4100	4.1	
194	B/o Gowsalya	LN	2.2	40 wks	Yes	Asymmetrical			<25	No							13.3	9700	3.25	
195	B/o Sharmila devi	LN	1.3	33 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/LBW		Yes		Yes		17.9	8400	1.36	Yes
196	B/o Swathi	LSCS	2	35 wks					<25	Yes	Preterm/LBW		Yes				21.9	12500	1.17	
197	B/o Mutheeswari	LSCS	2.5	36 wks						No							15.5	10300	2.28	
198	B/o Rathiselvi	LSCS	1.7	35 wks	Yes	Asymmetrical	Yes		<25	Yes	Preterm/LBW						16.5	6700	4.3	
199	B/o Roobiya devi	LSCS	2.2	36 wks					<25	No							16.7	5100	2.2	
200	B/o Kaatupoochi	LSCS	1.5	34 wks	Yes	Asymmetrical	Yes		<25	Yes	Preterm/LBW	Yes	Yes		Yes	Yes	17.3	17700	1.44	
201	B/o Priya	LSCS	2.5	37 wks						No							12.2	11700	2.25	
202	B/o Mahalakshmi	LN	2.4	38 wks	Yes	Asymmetrical			<25	No							16.3	14800	1.17	
203	B/o Bhuvaneshwari	LSCS	2	36 wks					<25	Yes	Resp distress						17.1	5400	2.23	
204	B/o Nandhini	LSCS	2.6	36 wks						No							16.9	9600	3.35	
205	B/o Sugantha	LSCS	2	35 wks						No							15.8	8100	1.83	
206	B/o Sandhana devi	LN	2.1	35 wks			Yes	Yes		Yes	Preterm/LBW				Yes		19.5	14500	0.73	
207	B/o Ayyammal	LSCS	2.3	39 wks	Yes	Asymmetrical			<25	No							11.2	6700	1.83	
208	B/o Thanyeswari	LSCS	2.4	36 wks						No							14.5	4500	2.65	
209	B/o Yogeswari	LSCS	2.4	37 wks	Yes	Asymmetrical			<25	No							18.3	12300	3.7	
210	B/o Karthiga	LSCS	2.3	36 wks						No							17.2	9600	1.16	
211	B/o Jothimani	LN	1.8	36 wks	Yes	Asymmetrical			<25	Yes	Resp distress						18.1	11300	0.9	
212	B/o Sathya Priya	LN	3	40 wks						No							17.9	5200	1.25	
213	B/o Suganthi	LSCS	2.4	41 wks	Yes	Asymmetrical			<25	No							18.1	6700	2.3	
214	B/o Vijayalakshmi	LSCS	2.6	40 wks					<25	No							17.7	12100	2.89	
215	B/o Muthulakshmi	LSCS	2.5	36 wks						No							16.2	6100	1.7	
216	B/o Lavanya	LN	1.9	34 wks			Yes		<25	Yes	Preterm/LBW	Yes	Yes				20.1	19600	1.32	
217	B/o Pandiammal	LSCS	2.5	36 wks						No							14.4	5800	1.9	
218	B/o Meena	LSCS	1.1	35 wks	Yes	Asymmetrical			<25	Yes	Preterm/IUGR				Yes		18.3	6700	4.21	
219	B/o Nagarathinam	LN	2.6	36 wks						No							16.5	3600	1.6	
220	B/o Priya	LN	1.6	34 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/LBW	Yes	Yes				14.8	12500	2.21	
221	B/o Dheebulakshmi	LN	2.4	40 wks	Yes	Asymmetrical			<25	No							16.6	9800	1.45	
222	B/o Nagajothi	LSCS	2.3	36 wks						No							16.1	15700	0.47	
223	B/o Sumithra	LN	2.4	37 wks	Yes	Asymmetrical			<25	No							15.8	16400	1.23	
224	B/o Anusiya	LSCS	1.4	34 wks	Yes	Symmetrical	Yes	Yes	<25	Yes	Preterm/IUGR						16.9	9500	2.13	
225	B/o Ramyadevi	LSCS	3.4	40 wks						No							16.1	11600	1.7	
226	B/o Muthumari	LSCS	1.9	35 wks					<25	Yes	Preterm/LBW		Yes				19.1	22100	1.15	Yes
227	B/o Shanthi	LN	3.6	40 wks						No							17.2	4600	2.13	
228	B/o Sumitha	LN	2.3	36 wks						No							16.8	9400	2.4	
229	B/o Murugeswari	LSCS	2.6	37 wks						No							16.6	8700	3.16	
230	B/o Priyanka	LSCS	2.3	36 wks						No							13.3	12300	0.74	
231	B/o Sugapriya	LN	1.4	33 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/LBW		Yes				16.9	8100	0.23	Yes
232	B/o Sivasakthi	LSCS	2	35 wks						Yes	Preterm/LBW		Yes		Yes		18.7	7400	1.7	Yes
233	B/o Karthiga devi	LN	2.5	36 wks						No							19.3	3200	2.2	
234	B/o Gomathy	LSCS	1.5	34 wks	Yes	Asymmetrical	Yes		<25	Yes	Preterm/IUGR	Yes			Yes		17.1	13000	0.86	
235	B/o Pandeeswari	LSCS	2.4	39 wks	Yes	Asymmetrical			<25	No							16.9	12600	3.97	
236	B/o Pavithra	LN	2.2	36 wks					<25	No							17.8	12100	4.3	
237	B/o Aishwarya	LN	2.1	37 wks	Yes	Asymmetrical			<25	No							16.4	6200	1.83	
238	B/o Nagalakshmi	LSCS	2.4	36 wks						No							16.9	7400	3.2	
239	B/o Alagusundari	LSCS	3.1	39 wks						No							17.6	8100	1.71	
240	B/o Selvi	LN	0.9	29 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/LBW	Yes	Yes		Yes		20.3	19100	1.5	
241	B/o Rajalakshmi	LSCS	3.3	40 wks						No							11.3	8200	2.33	
242	B/o Muthumari	LN	1.7	34 wks	Yes	Asymmetrical			<25	Yes	Preterm/LBW		Yes		Yes		14.2	17100	1.4	
243	B/o Nandhini	LSCS	2.4	36 wks						No							15.7	11200	2.1	
244	B/o Gowsalya	LSCS	2	36 wks			Yes		<25	Yes							16.1	17600	1.91	
245	B/o Lakshmi	LN	2.3	38 wks	Yes	Asymmetrical			<25	No							14.7	6500	1.8	
246	B/o Veeramani	LN	2.5	36 wks						No							15.3	9400	2.3	
247	B/o Prema	LN	1.4	35 wks	Yes	Asymmetrical	Yes	Yes	<25	Yes	Preterm/IUGR				Yes		16.2	12100	1.5	Yes
248	B/o Muthuselvi	LSCS	1.8	35 wks	Yes	Asymmetrical			<25	Yes	Preterm/IUGR						16.7	11800	1.15	Yes
249	B/o Nagajothi	LSCS	2.6	37 wks						No							17.1	9100	2.23	
250	B/o Eswari	LSCS	2.5	36 wks						No							14.9	6300	1.78	
251	B/o Jenifer Fathima	LN	1.5	33 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/LBW	Yes	Yes				15.2	8700	3.1	Yes
252	B/o Janani	LN	2.4	36 wks						No							14.6	11300	1.73	
253	B/o Kalpana devi	LSCS	2.3	38 wks	Yes	Asymmetrical			<25	No							16.1	4600	1.41	
254	B/o Latha	LSCS	2.1	36 wks					<25	Yes	Resp distress						17.9	8100	0.45	
255	B/o Alagumani	LSCS	2.4	39 wks	Yes	Asymmetrical			<25	No							15.3	7800	1.2	
256	B/o Manimegalai	LSCS	1.7	34 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/LBW						16.8	6300	2.23	
257	B/o Maheswari	LSCS	2.2	40 wks	Yes	Asymmetrical			<25	No							15.7	20200	3.5	
258	B/o Parkavi	LN	1.9	34 wks			Yes		<25	Yes	Preterm/LBW	Yes					15.9	9200	1.76	
259	B/o Jeyalakshmi	LSCS	2.5	36 wks						No							14.8	4700	2.1	
260	B/o Veeramani	LSCS	2.9	40 wks					<25	No							20.2	5100	4.23	
261	B/o Devi	LN	1.8	35 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/LBW						19.1	21900	1.29	Yes
262	B/o Madhanpriya	LSCS	2.2	36 wks					<25	Yes	Resp distress		Yes				15.3	11100	0.69	
263	B/o Devi	LSCS	2.5	36 wks						No							14.8	6700	1.71	
264	B/o Deepa	LSCS	2	35 wks					<25	Yes	Preterm/LBW		Yes		Yes		16.1	12300	2.23	
265	B/o Sudhapandi	LSCS	3.2	41 wks						No							16.9	8100	1.8	
266	B/o Chithradevi	LSCS	3	36 wks						No							15.7	14400	2.3	
267	B/o Soniya	LSCS	1.7	36 wks	Yes	Asymmetrical	Yes		<25	Yes	Resp distress		Yes				15.3	16100	0.86	Yes
268	B/o Shalini	LSCS	2.4	36 wks						No							12.7	9700	2.21	
269	B/o Narayani	LN	1.5	34 wks	Yes	Symmetrical	Yes	Yes	<25	Yes	Preterm/LBW		Yes		Yes		17.9	6800	1.01	
270	B/o Selvi	LSCS	2	35 wks					<25	Yes	Preterm/LBW	Yes					21.3	14900	1.3	Yes
271	B/o Jeyalakshmi	LSCS	2.3	36 wks						No							15.5	3100	2.25	
272	B/o Muthulakshmi	LSCS	2.5	36 wks						No							14.3	8500	3.16	
273	B/o Divya	LSCS	2.4	40 wks	Yes	Asymmetrical			<25	No							18.9	7100	2.2	
274	B/o Vijayalakshmi	LSCS	2.3	41 wks	Yes	Asymmetrical			<25	No							17.7	10200	1.9	
275	B/o Veeramani	LSCS	1.8	34 wks					<25	Yes	Preterm/LBW		Yes				16.5	9800	0.57	Yes
276	B/o Nagamani	LN	2.2	36 wks			Yes		<25	Yes	Asphyxia	Yes			Yes	Yes	14.3	6700	4.15	
277	B/o Sapna	LN	1.2	33 wks	Yes	Asymmetrical			<25	Yes	Preterm/IUGR		Yes		Yes		16.9	14100	1.32	
278	B/o Vaneeswari	LN	2.2	40 wks	Yes	Asymmetrical			<25	No							17.1	7100	1.8	
279	B/o Veerammal	LSCS	2	38 wks	Yes	Asymmetrical			<25	No							19.9	5800	2.23	
280	B/o Sangeetha	LSCS	1.8	36 wks	Yes	Symmetrical			<25	Yes	Resp distress		Yes				15.5	7200	1.4	
281	B/o Vinitha	LSCS	2.4	36 wks						No							16.3	6600	2.12	
282	B/o Roma	LN	2.6	37 wks						No							20.2	5400	1.12	
283	B/o Renukadevi	LSCS	1.7	35 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/IUGR	Yes					17.4	4300	0.83	
284	B/o Mariammal	LSCS	2.5	36 wks						No							18.1	11300	1.98	
285	B/o Chithra	LN	2	35 wks					<25	Yes	Preterm/LBW						17.5	21200	2.01	
286	B/o Amuthamala	LSCS	2.7	37 wks						No							16.1	9700	3.5	
287	B/o Veeralakshmi	LSCS	2.2	35 wks						Yes	Resp distress		Yes				14.7	14200	1.76	
288	B/o Rajeswari	LSCS	2.4	38 wks	Yes	Asymmetrical			<25	No							13.2	10500	1.9	
289	B/o Nagalakshmi	LN	2.3	36 wks						No							16.7	6600	1.2	
290	B/o Punitha	LN	2.4	36 wks						No							21.3	5100	0.89	
291	B/o Anitha	LN	2	40 wks	Yes	Asymmetrical			<25	No							16.4	8400	1.13	
292	B/o Vasantha	LN	1.6	34 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/LBW	Yes	Yes		Yes		15.8	18100	1.41	
293	B/o Kavitha	LSCS	2.3	39 wks	Yes	Asymmetrical			<25	No							15.5	3600	2.04	
294	B/o Nagalakshmi	LSCS	3.1	40 wks						No							16.1	7800	1.8	
295	B/o Muthuramya	LSCS	1.8	36 wks	Yes	Symmetrical			<25	Yes	Resp distress		Yes				17.7	6400	2.23	
296	B/o Kalaiselvi	LSCS	2.5	36 wks						No							16.4	11100	3.6	
297	B/o Lavanya	LSCS	3.6	40 wks						No							15.9	6100	1.4	
298	B/o Ranjani	LSCS	2.2	36 wks					<25	Yes	Resp distress		Yes				14.3	9700	2.1	
299	B/o Sudha	LN	1.7	34 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/LBW	Yes	Yes		Yes		17.7	8400	1.91	
300	B/o Priyadharshini	LSCS	3.2	39 wks						No							18.3	13600	2.02	
301	B/o Chellammal	LN	1.9	35 wks			Yes		<25	Yes	Preterm/LBW		Yes				16.1	11500	1.23	Yes
302	B/o Prabha	LSCS	2.4	39 wks	Yes	Asymmetrical			<25	No							15.9	9600	1.69	
303	B/o Jamuna	LSCS	1.8	37 wks	Yes	Asymmetrical			<25	Yes	IUGR						16.7	4500	0.86	
304	B/o Gayathri	LSCS	2	35 wks						Yes	Preterm/LBW		Yes				17.1	8700	1.45	
305	B/o Vinitha	LSCS	2.6	36 wks						No							19.6	6600	2.23	
306	B/o Prabha	LN	2.8	38 wks						No							18.2	11000	1.9	
307	B/o Nagajothy	LN	2.4	36 wks						No							16.9	14100	3.2	
308	B/o Muniammal	LSCS	1	30 wks	Yes	Symmetrical	Yes	Yes	<25	Yes	Preterm/LBW	Yes	Yes		Yes		17.7	9100	1.29	
309	B/o Lalitha	LSCS	2.5	36 wks						No							15.5	5400	2.8	
310	B/o Manimegalai	LSCS	3	39 wks						No							14.3	6300	1.6	
311	B/o Amutha	LN	1.2	33 wks	Yes	Symmetrical	Yes		<25	Yes	Preterm/IUGR	Yes	Yes		Yes		12.3	21200	0.56	Yes
312	B/o Philomena	LSCS	2.3	38 wks	Yes	Asymmetrical			<25	No							15.5	6700	2.6	